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LARYNGEAL OBSTRUCTION IN CHILDHOOD.[1]

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By way of introduction four differences between the larynx of the young child and that of the adult must be recorded: (i) the lumen is relatively smaller; (ii) the cartilaginous walls are softer and more easily distorted by the pull of the muscles; (iii) the soft tissues are looser and more vascular, allowing swelling to occur more easily as the result of inflammation or trauma; (iv) owing to the greater nervous instability of children, partial closure of the glottis from muscular spasm accompanies many laryngeal disorders and may dominate the clinical picture.

Part I: Non-Inflammatory Causes of Laryngeal Obstruction.

Congenital Laryngeal Stridor.

Congenital laryngeal stridor is due to the presence of an unusually flexible epiglottis, the lateral margins of which are rolled backward so that they almost meet posteriorly. The ary-epiglottic folds are thus brought close together, which gives the entrance to the larynx the appearance of being pinched from side to side. Congenital laryngeal stridor is not common. There have been only three cases at the Hospital for Sick Children in the past five years.

Greta H., a baby girl, aged twelve months, had been a "noisy breather" since birth. The noise varied somewhat from day to day and was worse when she was excited. Never a strong baby, she weighed only eleven and a half pounds on her admission to hospital. While she was in hospital slight chest recession was observed, together with fairly loud inspiratory stridor, which persisted day and night without causing distress. Expiration was quiet and her cry was not hoarse. There was no evidence of enlargement of the thymus or of any other mediastinal tumour. The laryngoscopic appearance was typical; each inspiratory effort was seen to cause an indrawing of the structures at the entrance to the larynx, the epiglottis moving downwards and the ary-epiglottic folds inwards. The vocal cords were normal.

General measures improved the child's health, and when she was examined six months later the stridor was less obvious, and with the laryngoscope only slight indrawing was seen to occur on inspiration. When she reported recently, at the age of two years, her breathing was noiseless and she seemed quite well.

This is a typical story. Congenital laryngeal stridor disappears spontaneously during the second year as the larynx enlarges and its walls stiffen. The prognosis is therefore good, except for the fact that these babies do not stand acute respiratory infections well.

Spasm of the Larynx.

Spontaneous attacks of laryngeal spasm in babies have earned the name of "*laryngismus stridulus*". Such attacks are one of the manifestations of infantile tetany, which itself is almost always a sign of rickets. The condition is chiefly of academic interest to us, because in Brisbane neither rickets nor *laryngismus stridulus* is seen.

The attacks occur in babies from six to twelve months of age. When mild they may consist merely of a series of crowing inspirations like the whoop of pertussis. In severe cases, however, sudden spasms of complete closure of the glottis may occur a dozen times a day. When this happens, the baby struggles violently, becomes blue and may lose consciousness or have a convulsion. A long crowing inspiration brings relief. Anti-rhachitic treatment will prevent a recurrence.

[1] Read at a meeting of the Queensland Branch of the British Medical Association on May 3, 1940, at Brisbane.

Foreign Bodies in the Larynx.

Aspiration of objects into the air passages is not an uncommon childish accident. Those which remain in the larynx tend to be of two distinct types: (i) large objects which stick in the entrance and cause rapid asphyxia; (ii) flat or irregular objects, which adhere to the walls of the larynx. After the first violent paroxysm of coughing there may be no particular respiratory distress, and all the symptoms of laryngitis may be present.

Smooth round objects, such as beads and peanuts, generally fall into the trachea.

Last year a boy, aged five years, suddenly choked while eating his dinner, struggled for breath and became cyanosed. He died just before he arrived at the hospital. At autopsy a piece of meat shaped like an inverted "V" was found in his throat, with one limb down the œsophagus and the other plugging the entrance to the larynx.

Another small child was admitted to the Adelaide Children's Hospital in 1937, with a history of chronic cough and hoarseness for the previous eighteen months. An X-ray picture of the chest accidentally included the larynx and revealed a small open safety-pin impacted there. It was so embedded in granulation tissue that its removal was difficult; but the child subsequently made a good recovery. The parents did not even remember the pin being inhaled.

Papilloma of the Larynx.

Papilloma is the only newgrowth of the larynx that occurs in children, and, even so, it is rare. There has not been a case at the Hospital for Sick Children in the past five years. The tumours, for they are usually multiple, are white wart-like growths, which may be attached to any part of the mucosal surface. They are thought to be caused by the virus which gives rise to the common warts of childhood, and, like the latter, they tend to retrogress at puberty.

Symptoms first appear in early childhood or late infancy, and at first suggest chronic simple laryngitis with hoarseness and a paroxysmal cough. Later, as the growths become slowly enlarged, the child suffers from the increasing obstruction of its airway and usually has attacks of paroxysmal dyspnœa.

The diagnosis of laryngeal papilloma can be made only with a laryngoscope, and the outlook is serious, for whether the tumours are removed surgically or destroyed by diathermy, they usually recur, and the vocal cords are apt to be damaged. I have no experience of radium therapy. Some surgeons adopt a conservative attitude and perform a tracheotomy when obstruction occurs. The tracheotomy tube is worn indefinitely in the hope that the papillomata will disappear at puberty.

Stenosis of the Larynx.

While not a primary condition, stenosis of the larynx must be mentioned in order to make the list of non-inflammatory causes of laryngeal obstruction complete.

The usual story is as follows. A child, generally a very young child, has an intubation performed for laryngeal diphtheria. The mucosa is eroded and ulceration follows in the region of the cricoid. The larynx becomes too irritable to retain the intubation tube, yet as soon as it is coughed out spasm of the glottis threatens to cause ashpyxia. Tracheotomy has to be performed and the cricoid ulceration heals, but the contracting scar tissue closes the lumen. In the worst cases the patient has to resign himself to wearing a tracheotomy tube for life.

Part II: Inflammatory Causes of Laryngeal Obstruction.

Acute Catarrhal Laryngitis.

Acute catarrhal laryngitis, a very common disease of the larynx, usually follows an infection in the naso-pharynx. It is particularly associated with the later stages of coryza and the early stages of measles. It is more prevalent during the winter months, and while it occurs at any age, it is most common between the ages of one and five years.

There is usually a fairly sudden onset of hoarseness and loss of voice, and the child has a croupy cough, which is often very irritating and always worse at night. On account of the smallness of the glottic opening, such obstructive symptoms as stridor, recession of the chest wall and even cyanosis readily appear in very young children.

The disease can generally be distinguished from laryngeal diphtheria by the absence of membrane and by the more rapid onset, and in severe cases by the higher temperature and flushed face. One is often deceived by the laryngitis of early measles, although Koplik's spots can usually be found at this stage if carefully sought.

An efficient steam tent is the most important part of the treatment, as the inhalation of warm, moist air soothes the inflamed mucosa and allays reflex spasm. A cough linctus is of value, and diphtheria antitoxin should be given if there is any doubt about the diagnosis. Surgical relief of the obstruction may occasionally be necessary, but I have not yet found it so.

Laryngitis Stridulosa.

Laryngitis stridulosa, which is closely related to acute catarrhal laryngitis, is characterized by nocturnal attacks of laryngeal spasm accompanied by dyspnœa and stridor. Nearly every case has an inflammatory basis, but occasionally attacks follow a digestive upset or exposure to cold. Children so afflicted are usually of excitable disposition and under six years old. Heredity may be a factor, but rickets plays no part. The attack may be preceded for several hours by slight hoarseness, or a barking cough in the evening may herald *laryngitis stridulosa* during the night.

In mild cases the dyspnœa caused by the laryngeal spasm may be insufficient to wake the child, but when it is severe he sits up in bed sweating, cyanosed and struggling for breath, with loud inspiratory stridor and chest recession. After an hour or more matters slowly improve, and during the next day, apart from hoarseness and a croupy cough, the child seems fairly well. There may be similar attacks for two or three nights in succession, or no more for several weeks.

Such severe attacks are very dramatic, but less common than the milder variety. Death during the seizure is almost unknown.

The best emergency treatment is to give an emetic and apply hot fomentations to the throat. An injection of adrenaline often helps to relieve the spasm. Phenobarbital (half a grain given at night for a child of two years) is the best safeguard against recurrence.

These attacks of laryngeal spasm, occurring as they do in association with catarrhal laryngitis, are closely analogous to the attacks of bronchial spasm (asthma) which in children generally occur in association with catarrhal bronchitis. Both are manifestations of involuntary muscle spasm produced in unstable children in response to an inflammatory change in the adjacent mucosa, and both are relieved by adrenaline, although *laryngitis stridulosa* less constantly so than asthma. Finally, children who suffer from *laryngitis stridulosa* in early life not infrequently develop asthma at a later stage.[1]

Syphilitic Laryngitis.

We see so little congenital syphilis in babies nowadays that syphilitic laryngitis is rare. It is usually an early symptom, occurring around three months of age, and it gives the infant a peculiar hoarse cry. There is seldom any serious obstruction to the airway, but ulceration occasionally follows. Diagnosis is not difficult, because other signs of congenital syphilis are present.

Œdema of the Glottis.

Œdema of the glottis may be produced by such diverse causes as a wasp sting on the back of the tongue, the impaction of a foreign body, acute inflammation in the pharynx or angioneurotic œdema. Perhaps the commonest single cause is the childish habit of putting the lips to the spout of a kettle to inhale the steam.

The epiglottis may be swollen to the thickness of a finger and can be seen if the tongue is depressed. The ary-epiglottic folds, which are out of sight, can easily be

felt as rounded swellings which tend to occlude the aperture of the larynx.

Even with a moderate degree of œdema inspiratory stridor and dyspnœa are present, and owing to the looseness and vascularity of the tissues in this region a dangerous degree of swelling can develop with great rapidity.

Some relief can be obtained by putting the child in a steam tent with ice chips to suck and an adrenaline spray (1 in 5,000) for the throat; but everything should be in readiness for the performance of tracheotomy at short notice if necessary. Intubation is inadvisable in the presence of œdema, because of the likelihood of trauma.

Retropharyngeal Abscess.

Retropharyngeal abscess, which causes laryngeal obstruction by pressure from without, is fortunately uncommon. I can recollect encountering only two cases in the past four years. It is particularly a disease of infants and may occur as early as the second week of life.

The primary infection is usually a streptococcal pharyngitis that spreads to the retropharyngeal lymph nodes, which lie on either side of the mid-line between the pharyngeal walls and the prevertebral muscles. If the lymph nodes are overwhelmed an abscess develops in this region.

A baby so affected is seriously ill, with a high temperature, vomiting or refusal of food, irritability and general prostration. Increasing pressure on the pharynx causes difficulty in swallowing and breathing, a hoarse croupy cough and a peculiar muffling of the voice. Respiratory obstruction may become severe enough to produce chest recession and cyanosis. At this stage the appearance of the infant is characteristic; he is flushed and restless and looks very ill; his breathing is noisy and the cervical glands are swollen. His mouth remains open and saliva dribbles, because it is difficult to swallow. His head is drawn far backwards to relieve pressure on the larynx and is inclined to the healthy side.

Diagnosis is not difficult, provided that inspection is made under a good light and is supplemented by digital examination. A soft round swelling can be seen and felt in the pharynx, slightly to one side of the mid-line, and its upper and lower limits can usually be defined. Occasionally the swelling is out of sight behind the larynx, but it is always capable of being felt. The abscess may push the posterior faucial pillar forwards and sometimes nearly fills the pharynx; but its situation is quite distinct from that of a peritonsillar abscess.

All writers stress the frequency of wrong diagnosis, the child dying perhaps during an attempted intubation. Without treatment either the patient dies of asphyxia or the abscess ruptures into the pharynx, in which case either spontaneous cure or fatal bronchopneumonia ensues.

The application of foments and the use of sulphanilamide are sufficient treatment in the early stages; but when a soft spot can be felt the abscess should be opened with a guarded scalpel. No anæsthetic is given and the head is kept low. The evacuation of pus gives immediate relief from dyspnœa and dysphagia, and, if care is taken to prevent its aspiration, the infant is cured.

Acute Laryngo-Tracheo-Bronchitis.

Acute laryngo-tracheo-bronchitis, a cumbersome word, denotes an alarming disease of childhood, which, while not particularly rare, has largely escaped recognition except in the United States of America. It may be defined as an acute descending infection of the upper portion of the respiratory tract, characterized by high fever and intense inflammatory change in the mucosa of the larynx, trachea and bronchi. Early swelling of the subglottic tissues produces respiratory obstruction severe enough to require surgical intervention for its relief. The mortality rate is in the vicinity of 50%. Life is threatened at first by toxæmia and later by asphyxia because of a sticky purulent exudate which plugs the trachea and bronchi. Even if the child surmounts these obstacles he generally contracts bronchopneumonia.

In its most severe form acute laryngo-tracheo-bronchitis is a fulminating disease, very much like the worst kind of laryngeal diphtheria. At the other end of the scale it blends imperceptibly with acute catarrhal laryngitis and tracheitis, and accordingly more than one writer has included in his series only those cases severe enough to require the introduction of a tube to prevent asphyxia.

No doubt it has been the difficulty of distinguishing acute laryngo-tracheo-bronchitis from laryngeal diphtheria that has caused it to be so frequently overlooked, for it is not rare. Brennemann[2] found 45 cases in ten years at the Chicago Children's Memorial Hospital, and Platou and Hilleboe[3] found 22 cases in twelve years at the Minneapolis General Hospital. In each instance it was responsible for about 6% of the total admissions for obstructive laryngitis. There were 15 cases at the Adelaide Children's Hospital between 1930 and 1938, and I have been able to find three among the records of the Hospital for Sick Children during the past five years. One was classified as "acute streptococcal laryngitis", one as "acute suppurative laryngitis", and the third as "œdema of the epiglottis; pneumonia, cardiac failure". All three children died.

Most cases are sporadic, but small epidemics occur from time to time, sometimes in association with epidemic influenza. Six of the Adelaide patients were admitted to hospital between January and July, 1938, and it is interesting to note that the admissions for acute catarrhal laryngitis, normally about 10 *per annum* at that hospital, jumped to 46 during the same seven-month period.

The disease is best described by two actual cases:

Mollie S., aged four years, was well until a few hours before her admission to hospital, when her breathing became noisy and difficult. She was admitted in considerable distress, with loud inspiratory stridor and chest recession. Her temperature was 100° F. She was placed in a steam tent and given 40,000 units of diphtheria antitoxin, but grew steadily worse. Tracheotomy had to be performed four hours after her admission to hospital, and gave immediate relief. The child remained comfortable all that day, but after twenty-four hours she again became distressed, with evidence of blocking below the tracheotomy tube. The temperature rose to 103·6° F., and she died the same day.

At autopsy the vocal cords and ventricular folds were greatly swollen, occluding the airway. The trachea and bronchi were congested and contained a quantity of thick yellow-green pus. There were a few petechial hæmorrhages in the lungs, but no consolidation.

Swabs taken from her throat on her admission to hospital, and material taken from the trachea at the time of operation and also at the autopsy failed to yield diphtheria bacilli and grew *Staphylococcus aureus* in pure culture.

Colin W., aged five years, had been ill for two days with stridor and difficult breathing, and his condition was so bad when he arrived that an emergency tracheotomy was performed in the casualty room. When he reached the ward half an hour later, his condition had improved considerably, and although his pulse was very rapid he was a good colour and was breathing freely through his tube. The tonsils were small and red, and there was an area of greyish exudate behind them and also on the posterior wall of the pharynx. The provisional diagnosis was laryngeal diphtheria.

He was given 40,000 units of diphtheria antitoxin and was treated in a steam tent, and for twenty-four hours he remained fairly comfortable. It was necessary, however, to clear his tracheotomy tube frequently, a fair amount of blood-stained mucus being obtained each time.

Next day his condition was much worse. His temperature was elevated and the muco-purulent exudate in the trachea had become less watery and more glutinous, so that his tube became blocked frequently. Trouble was also commencing lower down the trachea, so that the mere cleaning and replacing of his tracheotomy tube did not entirely relieve him.

For the next four days he hovered between life and death. The only way of relieving his respiratory obstruction was to remove his tracheotomy tube and pass a gum-elastic catheter—attached to a suction apparatus—down the trachea as far as the bifurcation. The lumen of the trachea was so much reduced that a number 10 catheter was a tight fit. A little sticky yellow pus and sometimes a few crusts were removed each time this was done, and partial relief was obtained for a few hours.

After four ghastly days his condition began to improve and he started to produce a thin, purulent, tracheal and

bronchial secretion which he could cough up unaided. The laryngeal obstruction was slow to subside, but he was able to do without his tracheotomy tube on the twelfth day. To my surprise he did not contract bronchopneumonia, and he was discharged from hospital at the end of five weeks with his wound healed and in fair health.

Swabs taken both from his throat and from the trachea failed to yield diphtheria bacilli, and, as in the other case, grew *Staphylococcus aureus* in pure culture.

It is natural that one should ask, in view of the unreliability of throat swabs, whether these cases described as laryngo-tracheo-bronchitis are not in fact cases of laryngeal diphtheria, the Klebs-Löffler bacilli not being found because of faulty technique or the overgrowth of secondary coccal invaders. This is a fair question; but there are several points against such a supposition:

1. In the cases I have encountered, every effort was made to prove the lesion to be diphtheria, and attempts at culture were made with material taken from the trachea at the time of operation as well as from the throat. Many of the American observers have gone further and submitted their patients to direct laryngoscopic examination on their admission to hospital, taking material for cultivation directly from the larynx, often within twenty-four hours of the onset of symptoms.

2. The clinical picture does differ from that of laryngeal diphtheria in one important respect: tracheotomy brings relief for about twenty-four hours only. After this time, as the infection spreads down the trachea, obstructive symptoms return and the child is again dangerously ill. Such an occurrence after tracheotomy for laryngeal diphtheria is most uncommon, but in laryngo-tracheo-bronchitis it is the usual story.

3. There is no response to the administration of diphtheria antitoxin. In laryngeal diphtheria the first day is the worst, and following an adequate dose of serum and the relief of asphyxia the temperature and pulse rate decline. But in the disease under consideration the child's condition becomes steadily worse for four or five days, the temperature mounting, and then he recovers gradually, if at all.

Charles B., aged two years, had been ill for one day before he was admitted to hospital. He was feverish and drowsy and had been vomiting, and his breathing had been difficult for six hours. He had a temperature of 101° F. when first seen, he was flushed and restless, and expiratory and inspiratory stridor was present. The throat was red and the epiglottis œdematous, but no membrane could be seen. He was treated as suffering from diphtheria, but examination of swabs failed to reveal diphtheria bacilli. His condition grew steadily worse, in spite of the administration of 60,000 units of antitoxin. On the third day his temperature was 103·6° F. and the respiratory embarrassment was increased. Thereafter the laryngeal obstruction became less pronounced, but bronchopneumonia developed. His temperature rose to 104·6° F. and he died six days after his admission to hospital.

4. The mortality rate of laryngo-tracheo-bronchitis is much higher than that of surgically treated laryngeal diphtheria—50% against 15% in the same hands.

5. The histological picture is somewhat different. True membrane is not found in laryngo-tracheo-bronchitis, and there is more acute inflammatory reaction in the submucosa than is usual in diphtheria.

6. The small epidemics of laryngo-tracheo-bronchitis which sometimes occur bear no relation to the prevalence of diphtheria at the time. Indeed, the disease seems to be most common in the large American cities, where the incidence of diphtheria is at its lowest.

We could claim that acute laryngo-tracheo-bronchitis was a specific disease with much more assurance, however, if a single species of organism were always found in the larynx and trachea. Unfortunately, at least six species have been isolated on different occasions. In the American reports (which form the bulk of the literature) hæmolytic streptococci predominate, although in Bradford and Leahy's[4] series of 28 cases the *Streptococcus viridans* was more common. Some American writers go so far as to describe the disease as "streptococcal laryngo-tracheo-bronchitis".

On the other hand, in each of Gardner's[5] three cases, which were among the earliest reported, *Staphylococcus aureus* was grown in pure culture. This has been the only organism recovered in many of the Australian cases, including the four reported by Mathew,[6] and in eight out of 11 cases investigated by Beare.[7] Among seven of the cases at the Adelaide Children's Hospital in which a search was made for the causal organism, staphylococci were found on six occasions, five times in pure culture and once in association with hæmolytic streptococci. In yet other instances pneumococci and influenza bacilli have been the only organisms isolated.

Two very recent cases further illustrate the bacteriological problem which this disease provides. The two children were cousins, who lived in the same house and were admitted to hospital within a few hours of one another.

Rosalie F., aged eigth months, had been "off colour" for four days and had experienced increasing difficulty in breathing for the past twelve hours. She was almost moribund on her admission to hospital, being cold, pale and cyanosed, with gross obstruction of her airway. Her attempts at inspiration produced a peculiar muffled stridor. The pharynx was inflamed but devoid of membrane. Immediate tracheotomy was performed and a quantity of viscid mucopus was coughed out of the trachea, with instant relief. After she had slept peacefully for about two hours, signs of respiratory obstruction returned and cyanosis followed. Tracheal suction with a catheter proved unavailing, and she died shortly afterwards. Autopsy revealed inflammation of the mucous membrane of the larynx, trachea and bronchi. Thick mucopus was present in the bronchi and both lungs were collapsed. There was no evidence of diphtheria or of bronchopneumonia. The predominant organism grown in culture from the trachea at the time of operation was Friedländer's bacillus.

Geoffrey F., aged seventeen months, came into hospital soon afterwards, with a temperature of 101° F., cough, hoarseness and stridor. His symptoms were much less severe and he recovered under conservative treatment without giving any cause for anxiety. He was regarded as suffering from acute catarrhal laryngitis, and it is reasonable to suppose that he was suffering in a mild form from the same infection as his cousin. Nevertheless, a swab taken from his larynx yielded only a mixed culture of pneumococci and *Micrococcus catarrhalis*.

The only possible way out of this bacteriological tangle is to postulate a filter-passing virus as the infecting agent and to regard any bacteria found in the trachea as secondary invaders. This is the only satisfactory explanation for the similarity of the clinical picture and pathological lesions in consecutive cases, despite the isolation of different organisms. It also explains such instances as one reported by Green and Miller,[8] in which a violent illness speedily brought about a child's death, although nothing more potent was isolated from the larynx and trachea than the *Streptococcus viridans* and the *Micrococcus catarrhalis*. There is scope for original research in this connexion.

This is not the place to describe the treatment in detail. Owing to the impossibility of distinction from laryngeal diphtheria, antitoxin will always be given. Whether a preliminary intubation is carried out or not, tracheal suction will be required later. Therefore, unless facilities for repeated bronchoscopy are available, tracheotomy should be performed, so that a gum-elastic catheter may be passed down the trachea through the wound and suction carried out by this route.

Laryngeal Diphtheria.

Although laryngeal diphtheria is still the commonest cause of laryngeal obstruction in childhood, there is reason to hope that in our lifetime we shall see it become a rare disease. Meanwhile, as its frequency diminishes, there is an increasing likelihood of the diagnosis being missed.

One should remember particularly that there are two forms of laryngeal diphtheria: (i) The secondary type, which is by far the most common, may occur at any age. Laryngeal symptoms are preceded by faucial infection, and their onset is characteristically slow and insidious. On examination membrane can be seen in the throat. (ii) Primary laryngeal diphtheria, occurring in less than 10% of all cases, is apt to cause more trouble in diagnosis.

It is especially found in fat-necked children aged under two years. The illness begins with a hard brassy cough, and spasm of the glottis plays a large part in the production of the early symptoms. As no abnormality can be seen in the throat, these cases are easily mistaken for acute catarrhal laryngitis ("croup"). Even the throat swab is unreliable and frequently fails to yield diphtheria bacilli.

Dawn R., aged two years, was recently admitted to the Hospital for Sick Children. She had been ill for six days, with "heavy breathing" and a croupy whistling cough, which had been partly relieved by inhalations. She was a bonny fat child, with inspiratory stridor, brassy cough and scattered rhonchi in the chest. No membrane could be seen in the throat. She was sent into hospital by a capable physician as suffering from "laryngitis, tonsillitis and acute bronchitis"; but she was given 20,000 units of diphtheria antiserum as a precautionary measure. A few hours later she had two severe attacks of respiratory obstruction, and tracheotomy had to be performed. Swabs from the throat and trachea yielded a heavy growth of diphtheria bacilli.

The child recovered, and the moral is clear: If in doubt about a case of laryngitis, give diphtheria antiserum and keep the patient under observation.

Diagnosis: The unreliability of throat swabs is notorious, and even when there is visible membrane several attempts may have to be made before a positive result is obtained. Without doubt many of these "false negatives" are due to careless swabbing. Unless one wears a headlight it takes three people to swab the throat of a young child: one to hold the victim, one to direct the light of a torch and a third to wield the tongue depressor and the swab stick. Manzullo's bedside diagnostic test, which is based on the fact that diphtheria bacilli blacken potassium tellurite, is only of limited application. Suspicious exudate in the throat is painted with 2% aqueous solution of potassium tellurite and inspected in ten minutes. If there is no change in appearance, one can say with a fair degree of assurance (about 90% accuracy) that the condition is not diphtheria. On the other hand, a positive result (that is, blackening of the exudate) is of no value, for certain organisms other than diphtheria bacilli can produce the same result.[9][10]

General Treatment: One's object in treating a child suffering from laryngeal diphtheria is twofold—to overcome the effects of the circulating toxin and to prevent asphyxia; but beyond saying that the first objective calls for complete rest and the administration of adequate amounts of antitoxin, fluids and glucose, I shall not discuss it here. The prevention of asphyxia is especially important because the heart is already under the damaging influence of circulating toxin. A child with a foreign body in the larynx can stand much more restriction of his airway with less risk, because his heart is sound. One cannot afford to see a diphtheria patient struggling for breath. While the obstructive symptoms are chiefly due to the presence of membrane and to swelling of the soft tissues, spasm of the glottis may also play a part, particularly in young children with primary laryngeal diphtheria. It can be relieved by putting the child in a steam tent, by coaxing him to lie relaxed and still, and by the administration of sedatives. The use of morphine is questionable. It soothes laryngeal spasm like a charm; but it also alleviates the distress of anoxæmia, and so may postpone necessary surgical intervention for several precious hours. Therefore, as it is seldom possible in any given case to say how much of the respiratory obstruction is due to spasm of the glottis and how much to other factors, I avoid morphine and rely upon such sedatives as potassium bromide and chloral.

Operative Treatment: It is always hard to decide whether the time for surgical intervention has come; but one should remember that these children die of cardiac failure and should not be allowed to strain themselves by any unnecessary struggling for breath. If every inspiration is an effort and causes recession of the soft parts of the thoracic cage, and if the child's condition is not improving and the pulse rate is rising, there is no point whatever in waiting for cyanosis, restlessness, pallor and sweating. These are the symptoms of dangerous anoxæmia and should not occur.

If it requires an operation to give the child an airway, should one perform intubation or tracheotomy? Whatever the cosmetic reasons against it, there are, under our conditions in Queensland, two convincing arguments in favour of tracheotomy. One concerns the training of house physicians and the other the relative safety of the two methods.

Even its supporters agree that intubation is inadvisable or impossible under certain conditions: (i) in babies under twelve months of age; (ii) when membrane is present in the trachea, causing obstruction below the larynx; (iii) when the child repeatedly coughs out his tube; (iv) in private homes and small country hospitals, in fact almost anywhere apart from the large metropolitan hospitals, because of the lack of skilled assistance should the intubation tube suddenly become blocked or be coughed out. On the other hand, tracheotomy can serve every type of case. The house physician, therefore, who does not learn how to perform this operation safely has wasted his time in the diphtheria wards. For, however dexterously he may manage an intubation, once he leaves hospital he will probably never perform it again. To learn the technique of tracheotomy he must frequently assist in its performance; operations on the still, bloodless cadaver are of little teaching value. But, as cases of laryngeal diphtheria are not too plentiful and his time in the diphtheria wards is short, how will he see enough tracheotomies unless they are constantly performed?

The second argument is a matter of statistics. At the Queen's Memorial Infectious Diseases Hospital, Fairfield, Scholes,[11] in a series of 735 intubations, had a mortality rate of 14%. At the Hospital for Sick Children we have always treated "intubation patients" and "tracheotomy patients" in the same ward under identical conditions. Since January 1, 1937, there have been 55 "intubation patients" with 15 deaths—a mortality rate of 27·3%. During the same period there have been 27 "tracheotomy patients", five of whom were children whom intubation had failed to relieve. Yet there have been only four deaths in this series—a mortality rate of 14·8%.

Three deductions from these figures seem justifiable: (i) Intubation at the Hospital for Sick Children is more risky than intubation at Fairfield, perhaps because of the relative inexperience of the medical or nursing staff, or for some other reason. (ii) Despite this handicap, tracheotomy at the Hospital for Sick Children is approximately as safe as intubation at Fairfield. (iii) Under identical conditions, tracheotomy is safer than intubation. (It is necessary to point out, to avoid misunderstanding, that with one exception our intubations were carried out by enthusiastic exponents of the method and that the one exception was among the children who recovered.) I have omitted from the above discussion, as misleading, Scholes's figure of 61·7% mortality rate in a series of 47 tracheotomies, because this operation was performed only when intubation was thought inadvisable or had failed. Three factors make tracheotomy the sounder procedure for general use: (i) it has to be performed only once: (ii) subsequently there is much less risk of sudden closure of the airway; (iii) it enables the lower part of the trachea to be kept clear by suction if necessary.

In the notes of intubation patients one frequently finds some such report as this:

> 8.30 p.m.: Tube suddenly blocked. Child struggling and cyanosed. Tube removed with some relief. 9 p.m.: Chest recession increasing. Re-intubated. Very pale afterwards.

Several children died of cardiac failure after one or more such incidents.

Tracheotomy and After-Treatment.

Except on occasions of extreme urgency the trachea should be opened below the isthmus of the thyreoid, and not above it; the sensitive cricoid region is thus avoided. Plentiful infiltration with "Novocain" makes the tissues partly translucent, and after the skin incision the whole operation is carried out by blunt dissection until the trachea is exposed. Veins are gently drawn aside instead of being divided, and there is practically no bleeding.

After the tube has been tied in position a wet acriflavine dressing is kept on the wound. A drop or two of sterile

saline solution is instilled every hour to keep the tracheal secretions moist, and the tube is kept clear of fragments of membrane and mucus by a small rubber catheter attached to a suction apparatus. Sulphapyridine may be given prophylactically to reduce the risk of bronchopneumonia, once the principal complication. The tracheotomy tube can usually be removed after seventy-two hours.

There have been no instances of retained tube, hæmorrhage, sepsis or bronchopneumonia among our 27 cases. Two children died of toxic heart failure in spite of a normal airway, and the other two had extensive membrane in the lower part of the trachea and lower bronchi, and could not be relieved.

Summary.

1. The various conditions which cause laryngeal obstruction in children have been described.
2. A more complete account has been given of acute laryngo-tracheo-bronchitis, a disease which is little known in Australia.
3. In the discussion of the operative treatment of laryngeal diphtheria, reasons have been advanced for considering tracheotomy the operation of choice.

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THE MICROTOMY OF THE EYE.[1]

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BECAUSE of the special technical methods required for the preparation of satisfactory microscopic sections of the eye, the investigation of ocular pathology is largely neglected. Modern methods, however, have robbed the process of celloidin embedding of most of its difficulty, and tissues may now be embedded in that medium nearly as rapidly as in paraffin and at least as easily.

Prompt and adequate fixation is essential. Pure formalin should not be used, as it causes great shrinkage of the chorioid and renders investigation of that structure impossible. Kolmer's solution is very satisfactory and is less difficult to handle than Zenker's. It is composed of four parts of a 5% aqueous solution of potassium bichromate, four parts of formalin (10%) and one part of glacial acetic acid. For the special investigation of the visual cells of warm-blooded animals a saturated solution of uranyl acetate and a 50% solution of tri-chlor-acetic acid, of each one part, may be added. In animals the best results are gained by injection of the whole animal immediately after it has been killed with coal gas. The solution will not keep and must be prepared just before use. The carefully enucleated human eye is immersed in a large quantity, at least 100 cubic centimetres, immediately after its removal. It is not necessary to open the eye, as this is likely to cause collapse and distortion of the globe. Post-mortem material may be dealt with by injection of the fixative into the vitreous with a fine hypodermic needle as soon after death as possible. This will help to prevent necrosis until the eye is removed and placed in the fixative. After twenty-four hours the globe is washed in running water for a further twenty-four hours. This step may be omitted, and dehydration may be commenced immediately through steps of 35%, 50%, 75%, 95% and 100% alcohol. It is an advantage to hurry the process of dehydration, as prolonged immersion in alcohol hardens the lens and adds difficulty to the cutting. Three hours in each concentration is sufficient, and this may be reduced to half an hour in the paraffin oven at 60° C. Before the embedding is begun the globe is placed for a similar period in a mixture of equal parts of absolute alcohol and ether. The globe may now be opened by the removal of two opposite lateral slices of sclera, chorioid and retina. In routine pathological work, time spent in sectioning is saved by freezing the globe before dehydrating, and cutting in any desired plane with a sharp thin-backed knife.

For the embedding three solutions of celloidin are required, of 10%, 25% and 50%. These are prepared by dissolving di-nitro-cellulose of 1/2 second viscosity in a solution of alcohol and ether. As the nitro-cellulose is delivered packed in alcohol, the use of the quantities shown in Table I will suffice. The 50% solution will take a few days to dissolve. The alcohol is added first and allowed to stand overnight. The ether is added the following day.

TABLE I.

	Concentration.		
	10%	25%	50%
Nitro-cellulose	40 grm.	100 grm.	200 grm.
Absolute alcohol	200 c.cm.	180 c.cm.	160 c.cm.
Ether	200 c.cm.	220 c.cm.	240 c.cm.

It is not necessary to use special histological nitro-cellulose. That used in commerce is inexpensive and easily obtained locally. The writer has noticed no fading of sections after eighteen months' use.

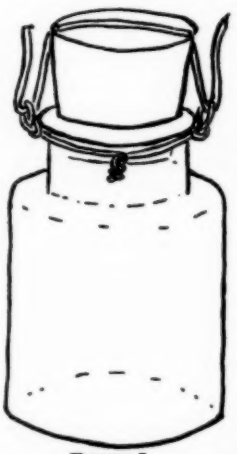

FIGURE I.

Heat is used to shorten the embedding time. A gas-tight bottle must be used and is prepared as follows (Figure I). Strong specimen bottles are selected, with no flaws in the glass and with a wide mouth. For the human eye bottles of 100 cubic centimetres' capacity are necessary, whilst those of 50 cubic centimetres' capacity may be used for smaller material. Large corks of good quality only must be used. A fresh cork is used for each embedding and should just fit into the mouth of the bottle. As it is removed and replaced several times it will sink more deeply into the mouth on each occasion. To secure the cork a piece of number 16 gauge soft-iron wire with two projecting ears is fixed under the flange of the neck of the bottle and held by twisting the ends firmly together. The specimen is placed in the bottle, covered with 10% solution of celloidin, and the cork is firmly inserted. The cork is fastened by passing a piece of number 16

[1] Work carried out with the aid of a grant from the National Health and Medical Research Council.

gauge copper wire through one ear, across the cork parallel with the cracks in it, and through the other ear. One end is looped over its ear and the other end is pulled tightly through the opposite ear and looped upwards. The bottle is placed in the paraffin oven at 60° C. for twelve hours, at the end of which period it is removed and allowed to cool slowly on a wooden bench, and the 10% solution is replaced with 25% solution. This is replaced with 50% solution after twelve hours. After a final twelve hours the block is ready for "casting".

The following precautions are necessary. An oven with an open flame must not be used. Do not attempt to cool the bottle suddenly under the tap, as it may crack and burst. Do not economize with corks and copper wire. The times are not critical, and may be shortened or lengthened according to the material used. The bottle may be left in the oven for weeks without harm.

The celloidin is hardened by immersion in chloroform, which need not be of the best quality. The specimen is placed in a paper "boat" covered with 50% solution of celloidin and placed in chloroform. It may be placed directly on a wooden or "Bakelite" block inside a paper receptacle formed by wrapping gummed paper around the sides of the block with its ends projecting. After an hour the paper is carefully peeled off. When the block sinks in the chloroform it is removed, and the block is rapidly trimmed to a cube. The material is transparent and orientation is easy. It is unnecessary to surround the tissue with an excess of celloidin. The smaller the block, the easier it will be to cut thin sections and to handle them. The block is removed to a second vessel of chloroform, and when it sinks in this it is ready for cutting. Excess celloidin solutions are poured back into stock and the chloroform may be used many times.

Small specimens may be scooped out with a spoon, with sufficient celloidin around them, and plunged straight into chloroform. A tough skin immediately forms on the celloidin, and if the specimen is not completely surrounded it may be pushed gently into place with a needle. As it hardens it is gently scraped off the spoon into the chloroform.

If an oven is not available or the tissue to be embedded is too large to be dealt with in this manner, it may be embedded by evaporation. The tissue is placed in 10% solution of celloidin in a Mason jar, which is tightly clamped and left for four days. The 10% solution is replaced by 25% solution for a further four days. After that time the specimen is placed in a stout paper "boat" and well covered with the 25% solution. It is replaced in the Mason jar, which is then closed, with the omission of the rubber sealing ring. As the solvent evaporates the celloidin will shrink. If the specimen is in danger of becoming exposed, more celloidin is added. After two days hardening is hastened by immersion in chloroform as before.

After the final hardening the block is ready for cutting. It will now be quite hard and transparent. It may be left indefinitely in the chloroform or stored in a mixture of equal parts of 90% alcohol and glycerine. Cut sections may also be stored in the same medium. Before insertion into the microtome the block must be mounted on a wooden, "Fibrolite" or "Bakelite" block. White pine is quite satisfactory and inexpensive, but must be well boiled before use to get rid of the resin. If "Fibrolite" is used, it must not remain immersed in chloroform for any length of time, as it will be ruined. To mount the celloidin block on the wood, the two clean opposing surfaces are painted with the alcohol-ether mixture, smeared liberally with 10% solution of celloidin and pressed gently together. The external surfaces of the joint are painted with chloroform and the complete block is stored in the alcohol-glycerine mixture until the joint is sufficiently firm. Small blocks may be cut in half an hour or less. It is wiser to allow large ones to stand overnight.

For cutting, a heavy microtome with a sliding knife is to be preferred. For large sections, one in which the actual cutting is done with knife and block totally immersed in spirit facilitates matters. There is a Jung microtome of this type, whilst less elaborate ones are made by Spencer, Bausch and Lomb, Leitz and Reichert. The writer uses the Jung microtome for large blocks and the Spencer microtome for smaller ones, although with the latter it is possible to cut sections at 12μ to 16μ of the whole human orbit. It is not necessary to have a special celloidin knife. A thin-backed flat-ground knife as recommended by Walls is excellent for the purpose. Whilst long knives are necessary for large blocks, it is better to use small ones for small blocks; they are much easier to sharpen, and a sharp edge is essential.

Practice is necessary before thin sections can be cut consistently. The block of tissue is inserted into the microtome, orientated as desired and firmly clamped. The knife is inserted into its holder with its cutting edge at an agle of approximately 15° to the horizontal and with its length at such an acute angle to its line of travel that almost the whole of the cutting edge traverses the block. It is then firmly clamped. A common cause of failure is lack of rigidity, either in the microtome or in the block of tissue. There must be no "play" in the moving parts of the machine; all adjustable parts must be tightly clamped, and all moving surfaces must be well oiled and moving freely. "Three-in-One" oil is perhaps the best to use. The celloidin block must be firmly attached to the wood, and often there is a little unsuspected movement here. If the embedding method outlined above is adhered to, there will be no lack of rigidity in the celloidin block. It is impossible to cut sections from one of india-rubber consistency, which slips away from the knife.

The beginner would be wise to commence with the eye of a guinea-pig and practice cutting at 10μ. During the cutting the block and the knife are kept flooded with 80% alcohol. The celloidin must be kept moist or it will shrink. The alcohol is applied with a camel's hair brush, which is also used to help the sections onto the knife. After the block has been levelled by several preliminary strokes of the knife, both block and knife are flooded and the brush saturated with alcohol is held over the block with the left hand, so that whilst not actually touching the surface it is held there by a fine film of fluid. The knife is pulled steadily across with the right hand, and as it crosses the block the left hand lifts the brush a little vertically. This action lifts the section onto the upper surface of the knife. If the edge of the brush is held directly over that part of the block first struck with the knife, the section will be prevented from rolling. The main action of the brush, however, is to provide a small reservoir of alcohol, which allows the section to slide up the knife without damage. Care must be taken not to get the brush under the cutting edge, or the latter may be damaged. With practice, and if this method is followed, a block the size of a guinea-pig's eye may be cut at 6μ at least. Sections at 16μ to 20μ may be cut without the brush and allowed to roll. They are not allowed to roll completely, but before the stroke is completed they are unrolled with the brush or the finger. It is not possible to manipulate thin sections in this manner. The section is removed from the knife by sweeping it down over the edge with the brush saturated with alcohol and transferred to a Petri dish of 80% alcohol.

The processes of staining, dehydrating and clearing are carried out in watch glasses or Petri dishes. If large numbers of sections are being dealt with at once, solutions may be pipetted on and off, or individual sections may be transferred from dish to dish with a section lifter or a needle. For any but the smallest sections a section lifter is to be preferred, one sufficiently large to hold the whole section without any part hanging over the edges. Changes from 90% alcohol to water and *vice versa* may be made without harm. Because of differences in surface tension, thin sections when transferred from alcohol to water "scoot" over the surface and may be damaged. This may be obviated by adding a trace of soap to the water. The pull of the surface tension may be used to unroll an obstinate curl in the edge of a section, by lowering the section, curled edge first, gently onto the water.

After Kolmer's fixative has been used, Weigert's iron hæmatoxylin gives good results. It is made freshly from equal parts of two stock solutions: (*a*) a solution of 1%

hæmatoxylin in 96% alcohol; (*b*) four cubic centimetres of *Liquor Ferri Sesquichloridi*, one cubic centimetre of officinal hydrochloric acid (specific gravity 1·124) and 95 cubic centimetres of water. Sections are placed in this solution for half an hour, rinsed in 90% alcohol and transferred to distilled water with a trace of sodium carbonate, where they are "blued" almost at once. After a quick wash in water they are placed in 90% alcohol before being counterstained with an alcoholic solution of eosin or phloxine. The latter is a splendid counter-stain for the visual cells, eosin being useless for this purpose. Fifteen to thirty seconds is sufficient. After being washed in 90% alcohol until no more stain is dissolved from the clear celloidin, the sections are dehydrated in a dish of absolute alcohol with a little chloroform added. The latter prevents the solvent action of the absolute alcohol on the celloidin, and must not be omitted. After a few seconds they are transferred carefully to the clearing fluid, where "scooting" again is apt to be troublesome. A mixture of nine parts of toluene and one part of terpineol is used for clearing. It may be used repeatedly and does not harden the sections. A Petri dish is used for the clearing fluid and a clean slide is placed in the bottom. A section is floated onto the slide with a needle and the slide is carefully lifted out and drained before a drop of benzol balsam is added and the coverslip is gently lowered into position. Instead of balsam, "Clarite", a new mounting medium, may be used. It is a synthetic resin dissolved in toluene ("Clarite" 60 parts by weight, toluene 40 parts), which remains neutral permanently, with the result that stains will not fade. It has a high melting point and will withstand the heat of a microprojector. A spring-type clothes peg, left on for twelve hours, will help to flatten the section.

Mallory's connective tissue stain is an excellent one for demonstrating the structure of the visual cells and for differentiating between the tissues of the ciliary region. Originally designed for use after fixation with Zenker's fluid, it gives just as good results after Kolmer's fixative, but will not stain correctly after formalin fixation unless special complicated methods are used. In its latest form it consists of two solutions: (i) 0·25% solution of acid fuchsin in distilled water; (ii) aniline blue (water-soluble), 0·5 gramme, orange G (85% to 90% dye content), 2·0 grammes, phosphotungstic acid, 1·0 gramme, and distilled water, 100 cubic centimetres. Sections are transferred from distilled water to the first solution, where they remain for half to one hour, and are then removed directly to the second solution for six hours at least. The times may be shortened if the staining dishes are placed in the embedding oven. After a rinse in water the sections are decolorized in 90% alcohol until no more stain leaves the periphery of the section. They are dehydrated, cleared and mounted in the usual manner. The thinner the section, the more brilliant the differentiation.

Additional Suggestions.

1. Care must be taken not to cut blocks containing bone or calcified tissue without first decalcifying them. Retinal "gliomata" are often calcified; and old, inflamed and degenerate eyes are often ossified. The globes of birds, lizards and turtles contain bony plates in the sclera which will work havoc with the knife. Kolmer's solution will decalcify tissue to a certain extent whilst it fixes it; but it should not be relied upon too much. Decalcification may be done after the embedding by placing the block in a solution of 10% nitric acid in 80% alcohol. With the tissues firmly supported in the celloidin there is no risk of damage or distortion.

2. Pigmented tissues may be bleached by placing the sections in 80% alcohol into which free chlorine has been bubbled from a flask containing a small quantity of strong hydrochloric acid and potassium chlorate crystals. This should be done in the fume cupboard. The sections should be well washed in alcohol for some days before being stained. Unfortunately they will not stain well with Mallory's stain after this process.

3. Sections stained by different methods may be mounted on the same slide. Several different staining methods may be conducted at once, and the sections may be collected in a common clearing dish and arranged on slides as desired.

4. Opaque stains, such as the second solution of Mallory, present a difficulty with small sections, which are easily lost. It is essential to pipette the stain off.

5. Cut sections may be stored indefinitely with the blocks in the storage fluid until required.

6. If very thin sections are required it is possible to cut down to 2μ by trimming away the block until a small piece of the required tissue is left. The edge of the knife must be perfect.

7. Absolute alcohol and ether dissolve celloidin; chloroform and water harden it. Hence sections must not be placed in absolute alcohol unless chloroform is added to it.

Summary.

The technique of celloidin embedding has been described with what might appear to be rather elaborate detail. As the process is radically different from the more commonly used paraffin technique, no apology is required for elaborating the minutiæ so essential for success. It has been said that the ocular pathologist is too much "wedded to celloidin" and that he is over-fascinated by the spectacle of a section of a whole eye mounted on a slide. Even with great care paraffin will not give the same results on ocular material, admittedly one of the most difficult to prepare satisfactorily. It is to be feared that such criticism often comes from sources unaware of the real difficulties and with no actual experience in celloidin embedding.

A large amount of the writer's experience was gained whilst working under a grant from the National Health and Medical Research Council. He wishes to thank Professor Wood Jones for granting him facilities for working in the anatomy department of the University of Melbourne and for giving him every help and encouragement. Dr. Andrew Brenan has kindly given him additional facilities in the pathological department of Saint Vincent's Hospital, Melbourne. The Spencer microtome is the property of the Ophthalmological Society of Australia. Finally, the writer wishes to record with gratitude the invaluable assistance in the details of fixation, embedding and staining so readily given by Mr. H. Marriott, of the Anatomy Department of the University of Melbourne, and by Dr. Gordon L. Walls, of the Ophthalmic Research Laboratory, Wayne University Medical School, Detroit, Michigan, United States of America.

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THE DIAGNOSIS OF MALIGNANT DISEASE.

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IT is well recognized that the depressing results of treatment in malignant disease can be enhanced in only two ways, namely, by improvements in therapeutic technique and by the earlier institution of treatment. The latter depends on earlier diagnosis, and in this paper an attempt is made to show wherein lies the failure to make this earlier diagnosis.

The basis of discussion is a series of nearly two hundred patients studied at the Austin Hospital, Melbourne.[1] A selection from the 100 cases with the most complete findings is presented in Table I; the other 100 cases are rejected because of probable unreliability of histories and insufficient proof of the conditions, and for other similar reasons. An effort was made to ascertain in every case the earliest symptoms and the lapse of time before advice was sought and treatment was given, and also whether the patient or the doctor was chiefly responsible when this time was unduly long. The conclusions arrived at will be stated later.

A few explanations are considered desirable before the reader is referred to the table. For example, the number of patients is too small to serve as a basis for generalizations, such as the usual symptomatology of any given condition. This, however, is not the object of the paper. Importance, too, should not be attached to the sex incidence, because on the one hand there were nearly three times as many male as female patients in the wards concerned, and on the other hand hardly any gynæcological patients are included.

The symptoms were learned chiefly from the patients concerned, but were in all cases taken or checked by myself with this investigation specifically in view. At times the histories could be checked to some extent, chiefly from the very brief notes supplied by the referring hospital at the time of the patient's admission, but at times by conversation with relatives. It is acknowledged, however, that many inaccuracies have probably occurred—we all know how difficult it is for even alert healthy people to remember occurrences and their dates, the difficulties associated with history-taking, and the unreliability of elderly people, especially of the hospital class. It is considered that the most important effect of this will be, in the present instance, to give an under-estimate of the alertness of medical practitioners, and apologies are extended to those unknown doctors who do not receive credit for their shrewd early diagnoses. At the same time, serious mistakes appear to have been made, and diagnoses have been missed even by those who, like myself, have had all facilities available. In this connexion the investigation showed that a diagnosis unsupported by autopsy is of no scientific value whatsoever; and moreover, an autopsy unsupported by microscopic examination of sections can be grossly misleading, as in Case VI of the second series, and in many prostatic cases. As Willis has stated:[1]

> For accurate scientific purposes the only mortality statistics worthy of serious consideration are those obtained from complete autopsy records with histological confirmation of the nature of all essential lesions present.

It is also necessary to enlarge on the meaning of "appropriate advice" in the sixth column of Table I. This is in most cases synonymous with "diagnosis", as most doctors refer patients to reliable colleagues or hospitals immediately. Occasionally the referee has acted less creditably than did the referring general practitioner. Sometimes full investigation or specific treatment for a known condition is contraindicated, and in such cases the institution of suitable general treatment has been considered as "appropriate advice".

Only the first one or two symptoms are given in some instances, as when the patient reports early, or when later symptoms have little bearing on diagnosis. The diagnoses given in the eleventh column were made in almost all cases at metropolitan public hospitals. Autopsies were performed (in the majority of cases by myself) except where the contrary is stated.

A study of Table I shows that, even if the histories are taken as reliable, three-quarters of the patients received "appropriate advice" within a month of first presenting themselves, but that about one-sixth had not received it within six months of this time. This latter figure indicates the existence of scope for improvement within the profession.

[1] A great deal of the information gathered by Dr. Oxer has had to be deleted owing to the restrictions on the use of paper.—Editor.

On the patients' side the figures are much worse. Very approximately, the numbers presenting themselves at various intervals were as follows:

Within one week	25%
Between one and four weeks	10%
Between one and six months	30%
Between six and twelve months	15%
Between one and two years	7%
Between two and three years	6%
Over three years	7%

In this small series the worst offenders, as groups, were patients with skin carcinomata (probably the most satisfactory lesions to treat) and women with breast tumours.

It is clear from these figures that education of the public will be necessary before much progress can be made in the treatment of these diseases. Ignorance or carelessness on the part of the medical profession is a comparatively small factor.

One or two points of interest arose in this investigation. One is the very great importance of the symptom "loss of weight" in the diagnosis of malignant disease. In nearly 10% of these cases it was the first symptom, and it was the second symptom in 20%.

A second point was the apparent complete localization, even at the time of death, in occasional cases of gastric carcinoma. Incidentally, the left supraclavicular gland, even when involved (as it often is in this condition), is frequently absolutely impalpable *ante mortem*. I do not recollect having noticed this point stressed in the text books.

We learn by our mistakes, and in the course of this investigation several more or less gross errors of diagnosis were encountered. These are presented in the second series of cases, and with one exception are additional to the cases quoted in Table I. The case histories given are self-explanatory.

Second Series.

In the following cases several more or less gross errors in diagnosis were made.

Case I.—A male patient, aged seventy-seven years, was admitted to hospital with a diagnosis of carcinoma of the prostate; he had a suprapubic cystostomy. The prostate was not malignant, but there was a ring carcinoma of the descending colon. The only symptom was diarrhœa, which commenced after his admission to the Austin Hospital.

Case II.—A male patient, aged sixty-five years, was admitted to hospital with a diagnosis of carcinoma of the prostate, with radiologically diagnosed secondary deposits in the spine. In the Austin Hospital lack of urinary symptoms led to reconsideration; but the diagnosis, even after further X-ray examinations of the spine, was not established until testicular swellings appeared. The diagnosis was actually tuberculosis of the spine, prostate and testes.

Case III.—A male patient, aged fifty-two years, was admitted to hospital with a diagnosis of carcinoma of the stomach. There was a well-defined simple ulcer of the stomach with carcinoma of the caecum. This case is referred to in Table I (Case XLI).

Case IV.—A male patient, aged seventy-three years, was admitted to hospital with a diagnosis of carcinoma of the larynx. He was suspected on his admission to hospital, on the history unsupported by obvious physical signs, to be suffering from pulmonary tuberculosis. There was a little sputum, which was found to contain tubercle bacilli. Lesions were found in the lungs at autopsy.

Case V.—A male patient, aged forty-five years, was admitted to hospital with a diagnosis of carcinoma of the rectum with secondary deposits in the pelvis; the diagnosis was based on biopsy and X-ray examinations.[2] Soon after his admission to hospital secondary deposits in the right lung were suspected and found radiologically. Autopsy disclosed hydatid of the pelvis, non-malignant stricture of the rectum, and non-malignant pulmonary abscesses.[1]

[1] I have just been informed by Dr. R. Kaye Scott that he has found evidence—overlooked by me in my search of the Royal Melbourne Hospital records of this patient—that the original rectal condition was a carcinoma. His anticipated further communication in this connexion is looked forward to with great interest, as indicating a complete cure by means of deep X-ray therapy. This would not, however, alter the contention that the patient's final condition was incorrectly diagnosed.

TABLE I.

Case Number.	Sex.	Age. (Yrs.)	First Symptom.	Period between First Symptom and Seeking Advice. (Weeks.)	Period before Appropriate Advice Received. (Weeks.)	Second Symptom.	Interval between First and Second Symptoms. (Weeks.)	Third Symptom.	Interval between First and Third Symptoms. (Weeks.)	Diagnosis on Admission to Hospital, and Basis.	Autopsy Report and Comments.
					Mouth, Tongue and Pharynx.						
III	M.	75	Ulcer on tongue.	*c.* 26[1]	*c.* 80	Enlarged cervical gland.	? 52[2]			Carcinoma of the tongue.	Carcinoma of the floor of the mouth. "Doctor said it was nothing."
V	M.	71	Pain from tooth rubbing tongue.	*c.* 16	16	Swelling of tongue.	*c.* 4	Pain, dysphonia, loss of weight.	*c.* 8	Carcinoma of the tongue.	No autopsy; diagnosis clinically certain.
					Œsophagus.						
XVI	M.	66	Weakness.	2	6	Anorexia.	4	Dysphagia.	6	Carcinoma of the œsophagus (X rays) and pulmonary tuberculosis.	Carcinoma of the œsophagus; hydatid disease of the liver (thought *ante mortem* to be malignant enlargement); pulmonary tuberculosis—had this right through the last war.
XX	M.	71	Dysphagia.	4	4	Loss of weight.	*c.* 10			Carcinoma of the œsophagus (X rays).	Carcinoma of the floor of the mouth noticed on admission to hospital; carcinoma of the œsophagus probably the original lesion — both similar microscopically.
					Stomach.						
XXV	M.	65	Alteration in type of chronic indigestion.	16	17	Loss of weight.	8	Vomiting and hæmatemesis.	16	Carcinoma of stomach (X rays, laparotomy).	Carcinoma of the stomach.
XXVII	F.	66	Abdominal pain after food.	*c.* 50	*c.* 50	Anæmia.	*c.* 10[1]	Weakness and loss of weight.	*c.* 15	Carcinoma of the stomach (laparotomy).	Carcinoma of the stomach; there was no apparent extension beyond the stomach a year after laparotomy.
XXIX	M.	68	Belching and anorexia.	1	1	Loss of weight.	4			Carcinoma of the stomach (X rays).	Carcinoma of the stomach. Six months before the patient said that his first symptom appeared his doctor sent him for a gastric X-ray examination; no abnormality revealed. Three months after first symptom appeared, lesion was revealed by X rays.
					Intestines.						
XXXV	M.	77	Dysuria.	½[3]	½	Acute retention of urine.	½[3]			Carcinoma of the prostate.	Carcinoma of the descending colon.[4]
XLI	M.	52	Hæmoptysis.			Pain.		Anæmia.		*Carcinoma ventriculi* (X rays).	Gastric ulcer; carcinoma of the caecum.[5]
					Rectum.						
XLII	M.	65	Loss of weight.	*c.* 35	*c.* 50	Intermittent diarrhœa.	10	Anæmia.	18	Carcinoma of the rectum (laparotomy).	Carcinoma of the rectum; treated for piles at a public hospital four months before laparotomy.
L	M.	67	Loss of weight.	*c.* 6	*c.* 6	Diarrhœa and abdominal pain.	*c.* 4			Carcinoma of the rectum.	Carcinoma of the rectum; treated for colitis for previous three years, not found *post mortem*.
					Sinuses et cetera.						
LV	M.	57	Pain on mastication.	½	½					Sarcoma of the left antrum.	Macroscopically, ? sarcoma, ? carcinoma; microscopically, epidermoid carcinoma.

[1] *c.* = *circa*. [2] ? = uncertainty as to the exact time. [3] ½ = less than one week. [4] Variable diarrhœa during the patient's last few months was the only symptom of the malignant condition; this was not investigated, because of the age and frailty of the patient. [5] See footnote, page 342. [6] See page 342. [7] See page 342. [8] See page 342.

[1] *c.* = *circa*. [2] ? = uncertainty as to the exact time. [3] ½ = less than one week. [4] Variable diarrhœa during the patient's last few months was the only symptom of the malignant condition; this was not investigated, because of the age and frailty of the patient. [5] See footnote, page 342. [6] See page 342. [7] See page 342. [8] See page 342.

TABLE I—*Continued*.

Case Number.	Sex.	Age. (Yrs.)	First Symptom.	Period between First Symptom and Seeking Advice. (Weeks.)	Period before Appropriate Advice Received. (Weeks.)	Second Symptom.	Interval between First and Second Symptoms. (Weeks.)	Third Symptom.	Interval between First and Third Symptoms. (Weeks.)	Diagnosis on Admission to Hospital, and Basis.	Autopsy Report and Comments.
					Larynx, Lung.						
LVI	M.	54	Husky voice.	12	12	Loss of weight.	50	Severe "asthma".	125	Carcinoma of the larynx (biopsy, X rays).	Intrinsic carcinoma of the larynx. Biopsy at the outset indicated chronic inflammation. Patient did not return for observation as instructed; pulmonary metastases two years later.
LVIII	M.	73	Loss of appetite and bowel irregularity.	26	26	Loss of weight.	16	Blood-tinged sputum and painful tightness in chest.	26	Carcinoma of the lung (X rays).	Carcinoma of the left lung.
LIX	M.	56	Pain in the right foot.	6	10	Anorexia, weakness, loss of weight.	2	Dyspnœa and cough.	3	Abdominal carcinomatosis with secondary deposits in the bones.	Carcinoma of the left upper bronchus.[6]
					Skin.						
LXVII	M.	69	Lump on the ear.	*c.* 104	*c.* 104					Epithelioma of the ear.	No autopsy. Correctly diagnosed by a country doctor, but incorrectly treated for four months at a base hospital.
LXIX	F.	55	(i) Rash about the left eye. (ii) Rash about the vulva.	? 25	? 25 ? 300					Rodent ulcer of the orbit.	(i) Rodent ulcer of the orbit. (ii) Epithelioma of the vulva, checked microscopically.[7]
LXX	M.	71	Crack in the palm of the right hand.	*c.* 156	*c.* 546	Itching hand.	*c.* 364	Lump in the right axilla.	*c.* 728	Epithelioma of the hand.	Epithelioma of the right hand.
					Breast.						
LXXV	F.	65	Lump in the right breast.	*c.* 300	*c.* 300	Loss of weight.	*c.* 50[1]	Pathological fracture.	*c.* 280	Carcinoma of the breast.	Carcinoma of the breast. Further pathological fractures and gross cachexia.
					Genito-urinary Tract.						
LXXXVI	M.	52	Pain in the back after a fall.	½	½	Pain in the right leg.	2	Return of back pain, loss of weight.	4	Carcinoma of the left kidney, secondary deposits in the spine (X rays).	Adenocarcinoma of the kidney. X-ray examination of the spine at onset revealed no abnormality; "positive" five weeks later.
LXXXIX	M.	55	Scalding on micturition.	*c.* 300	*c.* 300	Pain in the bladder.	*c.* 200	Hæmaturia.	*c.* 250	Carcinoma of the bladder (cystoscopy, biopsy).	Carcinoma of the bladder. Patient treated himself with "...'s Bladder Pills".
XCII	M.	71	(i) Hæmaturia. (ii) Ulcer on the lower lip for years.	*c.* 25	*c.* 25	Frequency of micturition.	*c.* 16	Loss of weight.	?	Carcinoma of the prostate; epithelioma of the lip.	Carcinoma of the prostate. The lip lesion was excised just before patient's admission to the Austin Hospital; no recurrence.[8]
XCVII	F.	60	Loss of weight; tiredness.	*c.* 4	*c.* 35	Uterine hæmorrhage.	35			Carcinoma of the uterus (biopsy, laparotomy).	Carcinoma of the *corpus uteri*. Patient under medical care from the outset.
					Other Sites.						
XCVIII	M.	76	Hoarseness.	3	3	Dysphagia.	1	Cervical swelling.	2	Extrinsic carcinoma of the larynx.	Carcinoma of the thyreoid; not diagnosed correctly *ante mortem*.
XCIX	F.	52	Pain in the back.	?	38	Pain in the right arm.	30	Fractured right arm.	36	Metastatic carcinoma.	Multiple myelomata; diagnosed radiologically after admission to the Austin Hospital.

CASE VI.—A male patient, aged eighty-one years, was admitted to hospital with a diagnosis of carcinoma of the rectum, with a history of eleven years. Autopsy revealed what appeared to be a perfectly typical ulcerating carcinoma of the rectum, with secondary deposits in the lungs. However, examination of sections revealed silicosis of the lungs, the rectal lesion being also purely inflammatory. These microscopic findings were kindly verified for me by Dr. Willis.

CASE VII.—A male patient, aged sixty-seven years, was admitted to hospital with a diagnosis of carcinoma of the lung, after a bronchoscopic and X-ray examination. Autopsy revealed a hydatid of the liver spreading to the right lung, with the production of an empyema cavity.

CASE VIII.—A male patient, aged seventy-five years, was admitted to hospital with a diagnosis of abdominal malignant disease. After some observation in the Austin Hospital he was thought to be suffering from uræmia. The blood urea level (124 milligrammes per 100 cubic centimetres) appeared to confirm this diagnosis. A post-mortem examination revealed a hydatid of the liver, with terminal bronchopneumonia.

Conclusion.

A selection from a series of 100 patients suffering from malignant disease is presented, with special reference to the early symptoms. It is shown that the failure of early diagnosis is in most cases the fault of the patients and not of the medical profession.

Earlier diagnosis by medical practitioners will be achieved by: (*a*) consideration in all cases of the possibility of malignant disease; (*b*) careful history-taking; and (*c*) avoidance of misinterpretation of actual physical signs, if present.

The importance is stressed of loss of weight as a frequent early symptom of malignant disease.

Acknowledgement.

I desire to thank Dr. A. M. Hutson, medical superintendent of the Austin Hospital (now Captain, Australian Army Medical Corps), for permission to publish this article; this permission was given on behalf of Wing Commander E. T. Cato, at present serving with the Royal Air Force, who was honorary surgeon in charge of these patients.

References.

(1) R. A. Willis: "Latent Primary Tumours", THE MEDICAL JOURNAL OF AUSTRALIA, May 30, 1931, page 653.

(2) G. M. Oxer: "A Proctological Problem", THE MEDICAL JOURNAL OF AUSTRALIA, April 6, 1940, page 483.

Footnote 5 (Table I).—Symptoms commenced with recurrent hæmatemesis fourteen years before death. From the outset the patient was in constant medical care in different semi-public hospitals. There was an attack of "dysentery", with hæmorrhage, six years after the onset. Four years later pain across the lower portion of the chest commenced, and later vomiting was noticed. Anæmia was noticed about the same time. Severe anæmia and abdominal pain were the chief late features. Repeated investigations during this latter period failed to give a correct diagnosis. These did not, however, include a complete X-ray examination of the bowel—another example of the danger of assumptions and of incomplete investigations. This patient's blood picture improved 50% with the exhibition (late as it was started) of antianæmic remedies; and it may be here stated that even in hopeless cases of late malignant disease the administration of iron and/or the parenteral administration of liver often gives more symptomatic relief than anything else except, of course, sedatives.

Footnote 6 (Table I).—This diagnosis afforded considerable satisfaction, as the pulmonary symptoms were extremely vague; and even after a skiagram, taken soon after the patient's admission to hospital, had revealed a shadow in the left lung, there was considerable dispute as to its significance. No general agreement had been reached even at the date of death. The foot lesion, the presenting symptom, was presumably a metastasis, but cast no radiographic shadow. The patient was submitted for examination by candidates for the second part of the examination for the degree of master of surgery; none of them suggested the correct diagnosis.

Footnote 7 (Table I).—The patient asserted that the vulval lesion was referred to by her at the specialist hospital where her eye was treated, but that no attention was paid to it.

Footnote 8 (Table I).—In these prostatic cases malignant disease was demonstrated by the occurrence of secondary deposits, or by microscopic examination of tissue, or in both ways.

THE TREATMENT OF MENINGITIS DUE TO THE MENINGOCOCCUS, HÆMOPHILUS INFLUENZÆ, PNEUMOCOCCUS AND STREPTOCOCCUS.

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THIS report deals with the patients suffering from purulent meningitis treated in the Royal Alexandra Hospital for Children since drugs of the sulphonamide group have been used. During this period there have been admitted to the hospital 25 patients with influenzal meningitis, 15 with meningococcal meningitis, 14 with pneumococcal meningitis, and five with streptococcal meningitis. Patients with meningitis secondary to a meningocele or to umbilical sepsis have not been included.

The mortality rate of meningococcal meningitis in the hospital has been reduced from 50% in the preceding ten years to 13·4%; that of influenzal meningitis from 98·4% in the preceding seven years to 64%; that of streptococcal meningitis from near 100% to 60%; and that of pneumococcal meningitis from 100% to 85·7%, even when deaths occurring within a few hours of the patients' admission to hospital are included. The two drugs we have used most extensively are "Prontosil album" (*p*-amino-phenylsulphonamide) and "M & B 693" (2-sulphanilylaminopyridine). Both drugs are given by mouth; if vomiting precludes this route their soluble derivatives may be given by intramuscular injection. Soluble "M & B 693" causes sloughing unless injected deeply into the muscles. The drugs are readily absorbed from the stomach and rapidly excreted by the kidney. The blood concentration reaches a maximum approximately three hours after oral administration. Renal excretion reaches a maximum less than one hour later, and by the end of a further two hours the blood concentration has fallen to half its maximum level. Therefore, to avoid gross fluctuation of the blood concentration, the drugs must be given every four hours, day and night. For most conditions in which this therapy is indicated in childhood, a dosage of one grain per pound of body weight per day, divided into equal doses given every four hours, is adequate. However, the concentration of the drugs in the cerebro-spinal fluid, while running parallel with the blood concentration, is only 50% to 70% as high. In so grave a disease as meningitis dosage should therefore be calculated on a basis of two grains per pound of body weight per day, and even this may be safely exceeded in most cases.

To ensure that the desired blood concentration is reached as rapidly as possible, it is worth while to make the initial dose double the calculated four-hourly dose, or even more. As the child's condition improves the dosage is diminished, but not too soon or too rapidly, and administration every four hours is maintained. The danger of reducing the dose too early or too severely is eloquently demonstrated by Cases XXI and XXIII of the influenzal series, in both of which relapse followed this false step.

Both drugs are well tolerated by children, especially young children. Cyanosis is common when "Prontosil" is used, but can be disregarded unless very pronounced. Depression of the level of circulating erythrocytes is the commonest toxic effect of "M & B 693", and granulopenia occurs occasionally. Either change appears to impair the resistance of the child and reduces the effectiveness of the drug. When a child is having "M & B 693" for more than a few days these changes should be sought by regular blood counts and controlled by blood transfusion. Vomiting is seldom seen in children. Skin rashes and hæmaturia occur occasionally.

Meningococcal Meningitis.

Meningitis due to the meningococcus is the least serious of all the forms of purulent meningitis. Various types of specific serum have been used with effect for a number of years and had reduced the general mortality rate of the endemic form of the disease to a figure of approximately 50%,[1][2] though much better results are claimed

by individual workers, on the one hand by the intravenous administration of massive doses of meningococcal antitoxin,[1] and on the other by the intrathecal use of anti-meningococcal serum.[2] The serum commonly in use in Australia is polyvalent and antibacterial.

During the ten years from 1928 to 1937 a total of 74 patients were treated in this hospital, and of them 37 died, 30 recovered and 7 recovered but developed sequelæ within a short time. Serum was given to most of these in various combinations of the intravenous, intramuscular and intrathecal routes.

Since the sulphonamide drugs have been used results have been very much improved, for of 15 patients treated only two have died, and both in rather special circumstances.

One was a mentally subnormal child, aged four months, with an extensive purpuric rash, and the other was a boy, aged seven years, with fulminating meningococcal septicæmia. Having given no reaction to the skin test for sensitivity to meningococcal antiserum, the boy was given an intravenous infusion of nine grains of sulphanilamide and 15 cubic centimetres of serum dissolved and diluted in 100 cubic centimetres of normal saline solution. During the injection severe collapse occurred and the boy died twelve hours later without having rallied. My feeling is that the therapy contributed to his death.

Table I summarizes the treatment of meningococcal meningitis. Most of the serum was given by intramuscular injection in equal daily doses. In Cases I, III, V and VIII the child received by intravenous injection a single dose of 10 to 60 cubic centimetres on the day of admission to hospital, and in Cases II and IV some serum also by intrathecal injection.

The table shows the striking decrease in the amount of serum given as our confidence in the sulphonamide drugs increased. "M & B 693" appears to be of more value than "Prontosil" in meningococcal infections. Usually the temperature fell greatly, the cerebro-spinal fluid cleared and the child's condition improved within three to six days; the drug dosage was then reduced to approximately half that stated. The four-hourly dosage was maintained, however.

The question arises as to whether "M & B 693" is so efficient in this disease that serum can be discarded. Exceedingly good results have been obtained with "M & B 693" alone; but the experimental work of Branham[3] suggests that a synergistic action exists between serum and sulphanilamide.

My present feeling is that the best treatment is vigorous therapy with "M & B 693". The rapid recovery in Case XV suggests that a dosage even higher than two grains per pound of body weight per day may be advisable. Serum is now discarded by many people, including the English Ministry of Health. Branham's work suggests that it may yet be found to have some place in the treatment of this disease. In the absence of "M & B 693" "Prontosil" is a valuable substitute. Daily lumbar puncture for the first few days should not be omitted.

Influenzal Meningitis.

Meningitis caused by the *Hæmophilus influenzæ* is a very much more serious disease than that caused by the meningococcus. The causal organism, a small Gram-negative bacillus, occurs sometimes in a coccal form, and the distinction between it and the meningococcus may be exceedingly difficult unless specific serological reactions are used.

A number of recoveries were reported before the use of drugs of the sulphonamide group and before the use of a specific serum. The bacteriological diagnosis must be questioned in such of these as depend on the morphology alone of the organism. All authorities, however, agreed that recovery was unusual. Wilkes-Weiss and Huntington[4] reviewed the literature and gave the death rate of children aged under two years as 97·6% and of persons aged over two years as 79·5%. In this hospital only one patient treated in the seven years before 1938 recovered—a mortality rate of 98·4%.

Since the beginning of 1938 at this hospital the disease has been treated in a variety of ways. "Prontosil", "M & B 693", a specific antiserum, human complement, hexamine and repeated spinal drainage have been used in various combinations. My impression of the value of each therapeutic agent has come not from tabulated results so much as from intimate association with each case.

The better to understand the results, two important factors in prognosis must be appreciated. The first is age. It has long been realized that older children were much more likely to recover than younger ones.[4][5] Of the children of this series who recovered, the average age was a little over seven years, and no child was less than two years old. Of those who died, the average age was a little over two years, nine patients (or 53%) were aged under two years, and the oldest was aged seven years. The ages of the children who recovered were: two and a half years, three years, five and a half years, six years, seven years, ten years, eleven years and thirteen years; the average age was seven and a quarter years. The ages of the children who died were: four months, six months, seven months, thirteen months (two), fifteen months, sixteen months, seventeen months, eighteen months, two years (two), two years and nine months, three years (two), three years and six months, five years, seven years; the average age was two years and two months.

Secondly, the other factors influencing the balance between infection and resistance of each patient must be considered. This involves such observations as the length of history, the general condition of the patient, the leucocytic reaction and the profusion of organisms in the cerebro-spinal fluid. Some attempt is made to record these observations in Tables II and III.

My impression of the value of each therapeutic agent used will be discussed in turn.

Table I.

Meningococcal Meningitis Treated with "Prontosil" or "M & B 693" and Meningococcal Antiserum.

Case Number.[1]	Patient's Age.	Duration of Illness Before Admission to Hospital. (Days.)	Total Serum Given. (Cubic centimetres.)	Duration of Serum Therapy.	Chemotherapy.		Duration of Chemotherapy.	Result.
					Preparation.	Grains per Day per Pound of Body Weight.		
	Yrs. Mths.							
I	7 0	1	15	One dose.	"Prontosil."	1·0	One dose.	Death.
II	0 4	5	30	One dose.	"Prontosil."	0·6	One dose.	Death.
III	3 6	1	240	6 days.	"Prontosil."	1·4	13 days.	Recovery.
IV	2 8	1	240	6 days.	"Prontosil."	1·6	22 days.	Recovery.
V	5 weeks.	10	130	15 days.	"Prontosil."	4·2-8·2	10 days.	Recovery.
VI	0 16	1	190	8 days.	"Prontosil."	2·1	7 days.	Recovery.
VII	3 10	3	250	10 days.	"Prontosil."	1·4	9 days.	Recovery.
VIII	0 10	21	80	4 days.	"Prontosil."	1·4	11 adys.	Recovery.
IX	0 8	7	70	7 days.	"Prontosil."	1·5	8 days.	Recovery.
X	0 11	3	60	3 days.	"Prontosil."	1·2	7 days.	Recovery.
XI	2 0	1	80	4 days.	"M & B 693."	1·4	12 days.	Recovery.
XII	1 0	2	60	3 days.	"M & B 693."	1·1	8 days.	Recovery.
XIII	13 0	2	30	3 days.	"M & B 693."	0·9	15 days.	Recovery.
XIV	0 4	3	20	1 day.	"M & B 693."	1·6	5 days.	Recovery.
XV	1 2	6	30	3 days.	"M & B 693."	4·0	3 days.	Recovery.

[1] In cases I, II, III, IV, VII and XIII a purpuric rash was present. The other children showed no rash.

TABLE II.

Influenzal Meningitis Treated with "Prontosil".

Case Number.	Child's Age.	Duration of Illness in Days before Admission to Hospital.	Examination of Smear from Cerebro-spinal Fluid.		Prognosis.	Dosage.[1]	Survival in Days after Admission to Hospital.
			Cells.	Organisms.			
	Yrs. Mths.						
I	0 6	8	+++	++	Very bad.	1·0	9
II	0 7	3	++	++	Very bad.	1·8	11
III	1 4	1	+++	+	Bad.	0·6	5
IV	13 0	2	+++	−	Good.	?	Recovery.
V[2]	11 0	6	+++	−	Good.	0·9	Recovery.
VI[2]	2 6	2	+++	Few.	Fair.	0·06	Recovery.
VII	7 0	2	+++	+++	Bad.	0·5	3
VIII	1 1	1	++	+++	Very bad.	1·0	1
IX[4]	5 7	1	+++	+++	Bad.	1·5	Recovery.
XI	0 4	7	++++	++	Very bad.	1·0	2
XII[3]	7 0	4	++	+	Good.	2·0	Recovery.
XIII	1 6	35	++	−	Very bad.	0·6	27
XIV	1 3	6	+++	++	Bad.	1·0	18
XV	3 6	1	−	++++	Very bad.	1·2	1
XVI	5 0	6	+++	+++	Bad.	2·3	4
XVIII[4]	2 0	3	+++	+++	Bad.	1·7	23

[1] Dosage is in grains per pound of body weight per day during the height of the illness.
[2] In Cases V and VI hexamine was given.
[3] In Case XII influenzal antiserum was given.
[4] In Cases IX and XVIII influenzal antiserum and human complement were given, and "M & B 693" was given during part of the illness.

TABLE III.

Influenzal Meningitis Treated with "M & B 693".[1]

Case Number.	Child's Age.	Duration of Illness in Days before Admission to Hospital.	Examination of Smear from Cerebro-spinal Fluid.		Prognosis.	Dosage.[2]	Survival in Days after Admission to Hospital.
			Cells.	Organisms.			
	Yrs. Mths.						
IX	5 7	1	+++	+++	Bad.	2·2–1·1	Recovery.
XVIII	2 0	3	+++	+++	Bad.	1·7	23
XIX	2 0	3	+++	+++	Bad.	1·7	25
XX	3 0	8 hours.	+++	Few.	Bad.	1·5	6
XXI	3 0	6	+++	++	Fair.	3·0–1·5	27
XXII	10 0	1	+++	Few.	Good.	1·5	Recovery.
XXIII	6 0	?	+++	+++	Bad.	1·6	Recovery.
XXV	3 0	1	+++	−	Fair.	3·0	Recovery.

[1] All patients had serum, and in Cases IX, XVIII, XIX, XX and XXI complement as well as "M & B 693". In Cases IX, XVIII and XXIII some "Prontosil" was given.
[2] Dosage is in grains per pound of body weight per day during the height of the illness. When two figures are given, they indicate a reduction in dosage after the first few days.

"Prontosil."

"Prontosil album" by mouth or "Prontosil soluble" by intramuscular injection was given to sixteen patients. A summary of the case records of these children is given in Table II. Of them, only five recovered, and in one of these cases (Case IX) the credit cannot be given solely to "Prontosil". In one other case (Case XII) the indefinite morphology of the organism and the rapid recovery suggest that the case was really one of meningococcal meningitis. The remaining three children who recovered had comparatively mild infections, and two of them were well up in years. Nevertheless, there is little doubt that they would have died without "Prontosil".

The condition of all the children in this group who died had a bad prognosis. Eight were under two years of age. Five had very severe infections, one indeed a hopelessly overwhelming infection (Case XV). Moreover, many of this group were treated in the comparatively early days of sulphanilamide therapy, and a survey of Table II shows that often the dosage was too small or too infrequent to maintain a constant and adequate concentration in the cerebro-spinal fluid.

When consideration is given to all these factors, the impression gained is that "Prontosil" has a definite value in the treatment of influenzal meningitis.

"M & B 693."

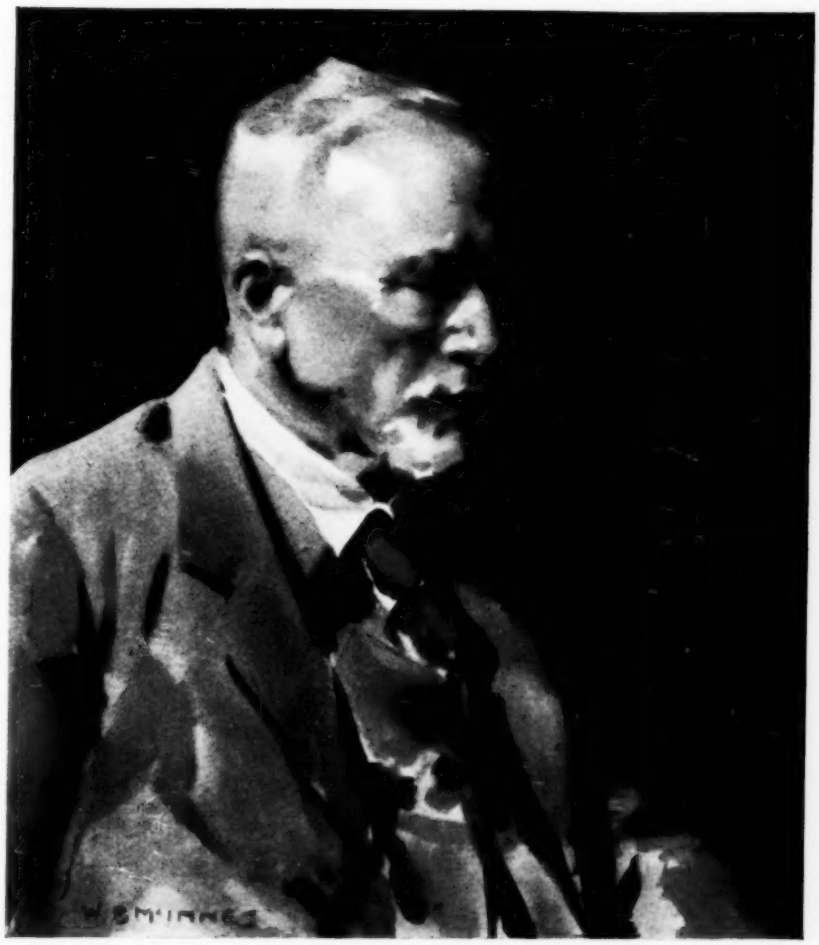
In eight cases "M & B 693" was used; four of these patients recovered (see Table III). The prognosis in this group, however, was distinctly better on the whole than in the group in which "Prontosil" was used. The group contained some children with severe infections who were very ill, but no very young children and no overwhelming infections. Moreover, the dosage of "M & B 693" was better regulated than in the earlier cases in which "Prontosil" was used; many of these later patients were energetically treated with serum. Indeed, Cases XXII and XXIII are the only ones in which "M & B 693" was unquestionably the mainstay of treatment.

The difficulty of assessing the comparative values of "Prontosil" and "M & B 693" from this series is therefore great. I incline toward the latter drug, for "Prontosil" given in Case XXII (when the boy was thought to have an œdematous pharyngo-laryngitis due to *Hæmophilus influenzæ*) failed to prevent the development of meningitis following tracheotomy; moreover "Prontosil", when given in Case XXIII, allowed a very distinct deterioration to take place in the child's condition, which had been kept stationary by "M & B 693".

There is no doubt that "M & B 693" is a valuable drug for the treatment of influenzal meningitis. It is probably more effective than "Prontosil"; but I feel that this question is not yet finally decided.

Influenzal Antiserum and Human Complement.

The use of specific antiserum in influenzal meningitis is theoretically sound. The various strains of *Hæmophilus influenzæ* which cause meningitis have very close antigenic relationships.[6] This does not apply to the respiratory strains of the organism, which exhibit a pronounced antigenic heterogeneity. Types of serum have been prepared against the meningitic strains, and, it must be admitted, have given disappointing results in the hands of very good workers.[4][6][7] However, Silverthorne[9] reduced the mortality rate from 98% to 72% by serum therapy. The difficulty of getting the serum into contact with the organism is a real one. In the early stages of the disease a septicæmia is present, and the intravenous or intramuscular injection of serum is rational. However, the



Arch^d. Watson.

amount of this serum passing from the blood to the cerebro-spinal fluid is distinctly limited. Intrathecal administration is more likely to be effective; but how far the serum spreads within the spinal theca is not known with certainty.

Ward and Fothergill,[8] in 1932, suggested that perhaps the reason for the failure of influenzal antiserum was the absence of complement from the cerebro-spinal fluid in this disease. The important role of complement in any antigen-antibody reaction has long been known, and many observers have noted its absence from the cerebro-spinal fluid in meningitis. Ward and Wright[9] also showed that *in vitro* complement was essential for the antibacterial effect of influenzal antiserum. They therefore recommended the intrathecal administration of complement in the form of fresh human serum as an adjunct to serum therapy. But still the results obtained were not good.

Sulphanilamide in our hands was probably of some value. Results obtained with "M & B 693" in other diseases gave reason for the hope that in this too it would prove of greater value. Serum and complement were theoretically and *in vitro* of undoubted value. It was therefore resolved to combine the three lines of therapy in practice. A high titre serum prepared against meningeal strains of *Hæmophilus influenzæ* was kindly provided by the Massachusetts Antitoxin Laboratory, Boston, Massachusetts.

In Case IX this combination was first tried. The child was severely ill, but recovered after a long illness. The method of treatment comprised oral chemotherapy, the intravenous administration of serum for the first few days, and the intrathecal administration of sensitized complement daily. The next two children to receive the serum by intravenous injection (Case X, and one in which the illness subsequently proved to be a pneumococcal infection) died rapidly of a toxic reaction following the serum injection. The fourth child (Case XII) recovered rapidly after oral chemotherapy and the intravenous administration of serum, but without the use of complement. After the serum, however, she had a severe toxic reaction, with rigors and high fever. The reactions in these three cases did not appear to be anaphylactic.

It was therefore resolved to give the serum by the intramuscular instead of by the intravenous route. The next child treated (Case XIV) was a very sick baby. Serum and "Prontosil", but no complement, were used. For some days the child showed definite improvement; but then the cerebro-spinal fluid became progressively more turbid and finally ceased to flow, and the child died. The next child (Case XVIII) had oral chemotherapy, the intramuscular administration of serum and the intrathecal administration of complement. For some days improvement took place, and then, when a relapse occurred, therapy unfortunately was not pursued as thoroughly as it might have been. In Case XIX the child had serum, complement and "M & B 693" and at first showed improvement, but then relapsed and lingered for many days. In Case XX similar therapy had little effect. The child was admittedly very ill on admission to hospital.

At this stage six patients had been treated since the arrival of the serum, four having had complement by intrathecal injection as well. Only two had recovered, and one of these (Case XII) possibly had a meningococcal infection. This was frankly disappointing and it was decided to give influenzal antiserum by the intrathecal route. In Case XXI the child received adequate oral chemotherapy, serum by intramuscular injection on the first two days, and serum and complement by the intrathecal route on most of the first eleven days. The child was then well and apyrexial, and the cerebro-spinal fluid was sterile. Unfortunately treatment was then drastically curtailed, the boy relapsed, and the disease could not be controlled again. I recognized the tendency of the disease to spontaneous remission and relapse, but felt that the boy would have been saved had he been treated more continuously.

In Cases XXII and XXIII two doses of serum were given by the intramuscular route with questionable effect.

In Case XXV vigorous chemotherapy and serum by the intramuscular route for three days and by intrathecal injection for six days were given, but no complement was administered. Steady improvement and ultimate recovery occurred.

Our present conclusions may be summarized as follows. Complement, given in the form of fresh human serum by intrathecal injection, seems to have been of little value. It possibly had the effect, perhaps only mechanical, of helping to maintain a free flow of thin cerebro-spinal fluid. One child of the five who received it recovered (Case IX) and all the others survived for a considerable period.

A specific influenzal antiserum does seem of some value, particularly if it is given by the intrathecal route. Serum given by the intravenous or intramuscular route is worth while probably only if the child is brought for treatment during the early days of his illness, when a septicæmia can be presumed or proved. At present I distinctly prefer the intramuscular route, the freedom from reaction outweighing the slight delay in absorption. In influenzal meningitis a further trial of the intrathecal route seems indicated. Only two children had serum by this route (Cases XXI and XXV) combined with "M & B 693" by mouth. One recovered without setback. The other became clinically well; his temperature became normal and his cerebro-spinal fluid free of organisms. When treatment was omitted the boy relapsed. "M & B 693" was then used vigorously again, but no serum was available for some days and the boy died. It is to be hoped that the Commonwealth Serum Laboratories will prepare a serum potent against the smooth meningeal strains of *Hæmophilus influenzæ* to facilitate such a trial.

The treatment I should now recommend for influenzal meningitis is large dosage of "M & B 693" by mouth and specific influenzal antiserum by intramuscular injection on the first day and by intrathecal injection each day until recovery is assured. It has been our practice to draw off as much cerebro-spinal fluid as possible at each lumbar puncture. The amount withdrawn should, of course, always exceed the amount of serum injected.

Pneumococcal Meningitis.

Pneumococcal meningitis does not respond to "M & B 693" in the striking way of pneumococcal pneumonia. The previous mortality rate was agreed to be almost 100%. A number of recoveries have been attributed both to sulphanilamide and to "M & B 693"; but a considerable number of cases are being reported in which the therapy has failed.

We have treated four patients with "Prontosil" and nine with "M & B 693" (Table IV). Of the former group, one child recovered, but developed hydrocephalus and died eight months later. In the latter group there were two recoveries.

TABLE IV.
Pneumococcal Meningitis.[1]

Case Number.	Age.	Chemotherapy.		Result.
		Preparation.	Dosage in Grains per Day per Pound of Body Weight.	
I	6 years.	"Prontosil album."	1·0–0·5	Death.
II	9 weeks.	"Prontosil album."	1·0	Recovery, but death from hydrocephalus eight months later.
III	5 years.	"Prontosil album."	0·75	Death.
IV	11 years.	"M & B 693."	1·3	Death.
V	16 months.	"M & B 693."	1·0–2·0	Death.
VI	7 years.	"M & B 693."	2·0	Death.
VII[2]	6 years.	"M & B 693."	2·2	Recovery.
VIII[2]	2 years.	"M & B 693."	3·5	Death.
IX	3 years.	"M & B 693."	1·4	Death.
X	13 years.	"M & B 693."	1·7	Recovery.
XI[2]	15 months.	"M & B 693."	1·4	Death.

[1] Cases in which death occurred within twelve hours of admission to hospital have been excluded from the table but not from the percentage mortality.
[2] Cases VII, VIII and XI had intramuscular injections of serum, prepared by the use of twenty-two types of pneumococci prevalent in Australia.

The type of pneumococcus was not determined in these cases, but this does not greatly alter the significance of the results. According to Fleming and his colleagues, the

susceptibility of pneumococci to "M & B 693" depends not on the type so much as on the individual strain of the organism.

It seems that vigorous administration of "M & B 693" is the most hopeful therapy yet available for pneumococcal meningitis. Nevertheless this remains the most lethal of the common forms of purulent meningitis.

Streptococcal Meningitis.

During the period under consideration four patients suffering from streptococcal meningitis have been treated and two have recovered (Table V). The bacteriological diagnosis of these cases is not satisfactory; a profuse growth of hæmolytic streptococci was obtained from the cerebro-spinal fluid of the child in Case II, but in all others the organism seen in the fluid resembled a streptococcus morphologically but failed to grow when culture was attempted. In Case IV the disease followed infection of the middle ear and mastoid cells.

Table V.
Streptococcal Meningitis.[1]

Case Number.	Age in Years.	Chemotherapy.		Result.
		Preparation.	Dosage in Grains per Day per Pound of Body Weight.	
I	12	"Prontosil album."	0·6	Recovery.
II[2]	8	"Prontosil album" (preceded by "M & B 693").	1·7	Recovery.
III	5½	"Prontosil album."	1·1	Death.
IV	2½	"Prontosil album."	1·5	Death.

[1] One case in which death occurred within twelve hours of admission to hospital has been excluded from the table but not from the percentage mortality.

[2] In Case II complement also was given by intrathecal injection, and before "Prontosil album" was given, "M & B 693", 1·7 grains per day per pound of body weight, was administered for two days.

It is worthy of note that the children who recovered were the older ones of the group. Chemotherapy was necessary for three weeks in Case I, but for only eight days in Case II.

In the hands of Silverthorne and Brown[10] sulphanilamide has reduced the mortality rate from hæmolytic streptococcal meningitis from 98·9% to 44·4%. There is no question of the value of this drug in this disease; but we have had no experience of "M & B 693". Until conclusive evidence is available to establish which of these drugs is the more effective in streptococcal infections, we prefer to use sulphanilamide.

Summary.

As a result of the treatment of purulent meningitis with drugs of the sulphonamide series the results have been improved very greatly in meningococcal meningitis, considerably in influenzal and streptococcal meningitis, and slightly in pneumococcal meningitis.

In meningococcal and influenzal meningitis immune serum has been used, with probable beneficial effect.

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Reviews.

INFECTIOUS DISEASES.

Anyone who writes a text-book on infectious diseases must find difficulty in setting limits to his subject. Dr. Harries and Dr. Mitman, doubtless making their selection from their own experience in fever hospitals, have omitted such highly infectious skin diseases as scabies and *impetigo contagiosa*; the venereal diseases; Asiatic cholera and plague; and, surprisingly, pulmonary tuberculosis. The last omission in particular gives the book[1] a sense of incompleteness, which is not in the least offset by the inclusion of a chapter on tetanus.

Nevertheless, from what they have chosen the authors have produced an excellent and attractive book. Over 100 pages are devoted to such general topics as infection and resistance, serum reactions, rashes, epidemiology and the control of infectious diseases in the community and in hospital. The common infectious diseases are described clearly and accurately, and there are good chapters on epidemic encephalitis, epidemic louse-borne diseases, infective jaundice, undulant fever, psittacosis and various infections of the alimentary tract.

All the relevant facts discovered in recent years have found their way into this section of the book, which is "full of good meat" and pleasantly served; and this should make it most valuable to the general practitioner. It contains enough material for M.R.C.P. and D.P.H. candidates, but rather too much for medical students, except those aspiring to an honours degree.

FRACTURES, DISLOCATIONS AND EPIPHYSEAL DISLOCATIONS.

In a "Manual of Fractures, Dislocations and Epiphyseal Separations" Harry C. W. S. de Brun sets out to write an "authoritative and concise work which leaves no perplexity in the mind of the physician who correlates adequate technical knowledge with sound common sense".[2] The book is a good one, but it consists of only 468 pages in an octavo volume. It is very doubtful whether a book of the type described can be written in anything under 700 pages. When a general practitioner wishes to consult a book on fractures, it is because he is in difficulty with a particular fracture, and in this case he requires all the details that he can find. A concise book will have no room for these details. The main criticism of this book is, therefore, that it is too much of a summary. When an author sets out to write a concise book it would be very much better if he confined it to the methods which he himself was accustomed to use. In this case the author is very fond of the method of traction with suspension. It would be better, therefore, if he described these methods alone and made no attempt to introduce descriptions of other methods which he does not use.

It is interesting to Australians to notice that the Hamilton Russell method of treating fractured femurs is apparently much used in the United States, and a full description of it is given; the author does not state that it is better used with both legs in suspension rather than with only the fractured leg. One minor criticism of the book is that although the author uses, as do all authors of good modern text-books, alterations in the type and numerals to indicate different points, he does not use them sufficiently or to the best advantage.

To sum up, the book is a good one, but it does not contain the details which are necessary for a general practitioner's work.

[1] "Clinical Practice in Infectious Diseases, for Students, Practitioners and Medical Officers", by E. H. R. Harries, M.D., M.R.C.P., D.P.H., and M. Mitman, M.D., M.R.C.P., D.P.H., D.M.R.E., with a foreword by W. A. Daley, M.D., F.R.C.P., D.P.H.; 1940. Edinburgh: E. and S. Livingstone. Demy 8vo, pp. 480, with illustrations. Price: 17s. 6d. net.

[2] "Manual of Fractures, Dislocations and Epiphyseal Separations", by H. C. W. S. de Brun, M.D., F.A.C.S.; 1940. Chicago: The Year Book Publishers Incorporated; Melbourne: W. Ramsay (Surgical) Proprietary Limited. Demy 8vo, pp. 468, with illustrations. Price: $3.50 net.

The Medical Journal of Australia

SATURDAY, OCTOBER 12, 1940.

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BLOOD TRANSFUSION SERVICES IN AUSTRALIA.

The history of the Spanish Civil War has shown beyond doubt, if indeed such proof was needed, that blood transfusion is a most important life-saving measure in modern warfare. Everyone will therefore agree that in the present crisis arrangements should be completed for the inauguration of blood transfusion services in the several States of this Commonwealth. Many Australians are still far too apt to look on Australia as secure in her southern isolation, secure against invasion, aerial bombardment and their appalling accompaniments. The reminder cannot be too frequent that when the superficial calm of this Commonwealth is disturbed the upheaval will be sudden and probably widespread. There will be no time then for conferences, reports, suggestions and plans that are so dear to certain persons; it will be too late then to say that we shall have a blood transfusion service and that it shall be arranged in such and such a way under the control of specified medical practitioners, who may possibly be more useful if allotted to other tasks. The result will be only muddle and inefficiency. For this reason we welcome a scheme, complete in every detail, that has been drawn up by a subcommittee of the New South Wales State Medical Coordination Committee. The subcommittee is composed of Lieutenant-Colonel A. M. McIntosh, Assistant Director of Medical Services, Eastern Command (chairman), Major E. F. Thomson (honorary secretary), Professor W. K. Inglis, Dr. F. B. Byrom, Dr. E. B. Jones and Dr. E. L. Morgan. There is no need to give readers a full outline of the details of this scheme. Much of the kind of information that it contains will be found in such a contribution as that of Dr. Ian J. Wood, published in this journal on November 18, 1939. Our object is to draw attention to, and to discuss, its more important aspects, and to urge the immediate adoption by the central authorities of them or of modifications of them, together with the other recommendations, either *in toto* or as part of some other plan of action.

It is proposed that an organization of voluntary blood donors be set up at once to cover Sydney and its suburbs. The organization recommended is to deal with present civil needs, but is to be capable of immediate adaptability in the event of a national emergency to cover both military and civil requirements. The service, which, it is held, should be controlled by an advisory committee, is to provide a twenty-four hour a day service of donors to hospitals and private practitioners. The subcommittee puts forward four suggestions regarding the administration of the service: from a central independent office, from the Department of Public Health, from a suitable public hospital, from the Central District Ambulance. It also states that similar organizations, affiliated with the central service, should be promoted in country districts which it names. The first point about this suggested service, and one of the most important, is that it would include within its scope a number of voluntary organizations at present in existence. While no one will wish to damp the enthusiasm or check the efforts of willing workers in the public good, everyone will agree that overlapping of effort should be prevented and that the best possible use should be made of all who are available for this kind of work. Conceivably a central body might coordinate the activities of individual organizations and allow them to retain their identity, at the same time creating new centres of activities where none existed. Apart from the fact that individual variations in methods of working in neighbouring districts or different organizations in the same district might lead to confusion, no one will deny that such concessions to self-regarding small bodies would be much less effective than would a single body acting for a whole State, provided the single body was administered with efficiency and understanding. The next question that arises is whether donors should or should not be paid. Services have been and are being run successfully with both paid and non-paid donors. In this instance the subcommittee thinks that the blood donors should be voluntary. We think that it is most important to keep alive in the community the idea of voluntary service, and are therefore inclined to agree that if voluntary donors can be procured they should be used, but only so long as the service covers the needs of the civilian population. If a state of emergency should arise within the boundaries of Australia itself, we should imagine that all medical services would be put under the control of one central authority. In these circumstances, and even if the needs of the civilian population are regarded as distinct from the military requirements and civilian medical services are controlled by one civilian authority, many more donors will probably be required than at present; most essential services will be paid for, and we think that the giving of blood should be looked on as a national service to be paid for by the nation. Out of this arises the question of the control of the service at its establishment. The subcommittee whose four suggestions have been mentioned favours the establishment of a central independent office. They adopt this view on the ground that a central independent office is more likely to be successful when divorced from the control of a government office or corporate body. We are doubtful of the wisdom of this decision. In the first place there is no evidence that a department of health cannot control such a service. On the contrary, though it pays its donors, nothing but the highest praise has been heard of the blood transfusion service run by the Department of Public Health of New

South Wales in connexion with its efforts to secure an improved maternity service (see THE MEDICAL JOURNAL OF AUSTRALIA, February 4, 1939). Again, the inauguration of a blood transfusion service will necessitate a certain amount of expenditure; this expenditure should be kept as low as possible and we should imagine that it will cost more to create a new and independent organization, with all its accessories of office and equipment, than it will to expand a service that probably exists already in the Department of Health. The argument that the efficiency of a blood transfusion service depends largely on the personal attributes of the officer in charge applies with equal cogency to an independent organization and to one controlled by a health department or corporate body. There is also the further consideration that the proposed service will in certain circumstances be reformed to meet both civilian and military needs. If this happens, the health department may be the authority charged with the supervision of civilian needs, acting, perhaps in liaison with or at the instructions of the military authorities; in these circumstances it would merely have to expand one of its own activities. If the health department was not so charged it would probably be accepted by the responsible authority as qualified to superintend a blood transfusion service more readily or at least as readily as any other body. Finally, consideration must be given to the legal position of a medical officer or other office bearer of a non-corporate body in the event of a legal action for damages in respect of a therapeutic disaster or a happening claimed so to be. Since a non-corporate body cannot be sued for damages, the officers of the organization would have to act as personal defendants in any such action.

In addition to those mentioned, the subcommittee discusses many other aspects. For example, for the collection of blood samples on a large scale, it recommends that the Deputy Director of Medical Services, Eastern Command, be asked to make available the organization used for the typing of blood of members of the Australian Imperial Force and that collecting teams be formed, consisting of medical officers with assistants from an organization such as the Voluntary Aid Detachment. Details of technique and of apparatus are given and a first-class scheme for the recording of transfusions and so on is included. In fact, little, if anything, has been omitted which would require consideration. Presumably when the New South Wales State Coordination Committee has considered this document, it will send it on with recommendations or emendations to the Central Coordination Committee. It is to be hoped that action will not stop at this point. The central authorities may have all the details for a blood transfusion service such as this complete to the minutest detail. If they have, the medical profession has not been taken into their confidence. There is no doubt that the medical profession and the public are growing impatient and want to be assured not only that a scheme is ready, but that it is working in a small and practical way among the civilian population, ready for expansion into a measure suitable for a national emergency involving the whole country. The subcommittee of the New South Wales State Coordination Committee has probably started a work of great national importance.

Current Comment.

EFFECTIVE DIPHTHERIA IMMUNIZATION.

IMMUNIZATION against diphtheria, by one or other of the approved methods, unquestionably increases the resistance of the patient to the disease. The immunity established is, however, by no means complete in all cases, as shown by a large number of reports of diphtheria occurring in immunized children. The best method for the evaluation of the procedure is undoubtedly the comparison of the incidence of the disease in a large immunized group and in an equally large non-immunized control group, as pointed out by H. P. G. Seckel.[1] The children of the two groups should be of the same age, social stratum and district, and should be observed at the same time. With this method it has been proved that the incidence of the disease in immunized children is only one-third to one-eighth or an even smaller fraction of that in non-immunized children.

Another statistical approach to the question is provided by the comparison of the incidence of the disease in any district before and after the introduction of large-scale immunization. To give information of any value such a comparison must cover long periods, for the incidence of diphtheria in any one district varies greatly from year to year, and waves of diphtheria morbidity recur regularly. In New York such a wave recurs each six and a half years, and was expected in 1934. But large-scale immunization of pre-school children had been commenced in America in 1929, and the expected wave of the disease in 1934 did not occur.

In general the severity of the disease in immunized children is less than in the non-immunized. Seckel has exhaustively reviewed and statistically analysed the type of disease reported in the medical literature as occurring in immunized children, and has compared it with the disease in a large non-immunized group. He found in the immunized group an overwhelming majority of cases of localized pharyngeal diphtheria and an almost complete disappearance of laryngeal diphtheria. The incidence of the toxic or malignant form of the disease was, however, about the same in both groups.

A variety of immunizing agents has been used to prevent diphtheria. In 1894 von Behring introduced antitoxin therapy. It so happened that a severe wave of diphtheria was waning in Europe that year, and the lower incidence of the disease in the next few years was wrongly and illogically attributed to von Behring's historic discovery. It is an excellent example of the comparison of disease incidence over too short a period and without reference to the waves of incidence of that disease—a method of evaluation condemned above. The falsity of these hopes was soon plain. In 1913 von Behring introduced immunization as a preventive of diphtheria by using a toxin-antitoxin mixture. In 1923 Ramon, of the Pasteur Institute, introduced anatoxin, or toxoid, a preparation which contained no toxin or animal serum and which seemed to give even greater protection than the toxin-antitoxin mixture. A series of injections, usually three, of each of these preparations is required. In 1932 the use of alum-precipitated toxoid began to supplant the use of these earlier preparations. A single injection seemed to produce rapid immunity, and without the unpleasant reactions so often encountered with other preparations. Varied reports on the reliability of a single injection of alum-precipitated toxoid are summarized by William B. Nevius and Ada C. McGrath.[2] In several large groups of children 89% to 95% of the children failed to react to the Schick test or had an adequate titre of antitoxin in the blood two years after immunization, results quite as good as those achieved by other methods. However, certain reports are summarized in which the results with alum-precipitated toxoid are greatly inferior

[1] *American Journal of Diseases of Children*, September, 1939.
[2] *American Journal of Diseases of Children*, June, 1940.

to those achieved by the use of plain toxoid or toxin-antitoxin. Thus Fraser and Halpern, of Toronto, gave one cubic centimetre of alum-precipitated toxoid to a group of forty children, and three one cubic centimetre doses of plain toxoid to thirty-five children. In ten weeks the antitoxic titre of the blood exceeded $^1/_{100}$ unit of antitoxin per hundred cubic centimetres of blood in 62% of the first group and in 91% of the second. After one year the figures were 19% and 91% respectively, showing a rapid loss of immunity when the alum-precipitated toxoid was used. Park reports results comparing the use of three one cubic centimetre doses of toxin-antitoxin, two one cubic centimetre doses of plain toxoid, and a single one cubic centimetre dose of alum-precipitated toxoid. With the first two methods a negative response to the Schick test was obtained at intervals of from one to three years in 93% to 100% of the children. In the group given alum-precipitated toxoid a negative response to the Schick test was obtained in only 60% of the children after one to two years, and only 18% of them after two to three years. Nevius and McGrath themselves report results after five years in two comparable groups of children. Seventy-eight children were given three one cubic centimetre injections of toxin-antitoxin, and a negative response to the Schick test was obtained in every instance after one year and in 96% after five years. Seventy-two children were given a single one cubic centimetre injection of alum-precipitated toxoid. A negative response to the Schick test was given by all of them after one year, but by only 80% after five years.

Despite the inconstant results of alum-precipitated toxoid, the authors still advocate its use because of the freedom from reaction and the ease of administration. They also recommend, however, that the Schick test should be applied to all children six months, three years and five years after immunization, and in the event of a positive reaction being obtained, that a further one cubic centimetre injection of alum-precipitated toxoid should be administered. The frequent impossibility of such a follow-up, even in private practice, detracts from the value of their routine practice and leaves the inclination to favour the use of plain toxoid or toxin-antitoxin mixture.

THE DEMONSTRATION OF TUBERCLE BACILLI IN THE URINE.

The usual process by which tubercle bacilli are sought in specimens of urine by clinical bacteriologists in this country consists of allowing the specimens to stand that sedimentation may take place, removal of the supernatant liquid, centrifugation of the remainder and the preparation of smears from the deposit. The difficulty of establishing bacteriological diagnosis in tuberculous disease of the urinary tract, even when cultural methods or animal inoculation is employed, is well appreciated. Interest therefore has attached to fruitful researches into the subject by J. H. Hanks and H. A. Feldman, of Washington, reported during the last two years.

Last year[1] these writers claimed to have shown that tubercle bacilli cannot be collected from urine efficiently by centrifugation alone. They have found from experiments with artificially infected urine that centrifugation can collect 99% of typhoid bacilli present in a given specimen, but only 20% of tubercle bacilli. They have also shown that when a flocculent precipitate is made to appear in the specimen of urine, as by suitable adjustment of the hydrogen ion concentration, the bacillary content of the rather bulky sediment resulting is very much greater, volume for volume, than in the very slight sediments obtained by direct centrifugation. "For purposes of guinea-pig inoculation or of cultivation", they write, "it should be noted that the total bacillary content of urate sediments exceeds those from centrifugation approximately one hundred times." In their most recent communication,[1] Hanks and Feldman report that they have studied the relative efficiency of several flocculation methods in the collection and concentration of tubercle bacilli. They have concluded that the production of "phosphate" flocculation, that is to say, of the flocculent precipitation of solids resulting from neutralization or slight alkalinization of urine with constant shaking, is a more efficient means of collecting the bacilli than the production of "urate" flocculation; and in addition it is more easily carried out and does not necessitate chilling of the urine, while the sediment is more constant in volume. The most uniformly efficient method for the collection of tubercle bacilli from the urine is to bring the sample to the "minimal flocculation point" by the addition little by little of sodium hydroxide solution with constant agitation.

In their earlier paper Hanks and Feldman asserted that to wash or to dissolve and centrifuge again a sediment once collected by any method resulted in a great diminution in the number of bacilli collected. However, in the phosphate flocculation method they have been able to devise a way of further concentration of the bacilli collected (and of provision at the same time of a means for their treatment with acid prior to cultivation) by dissolving the primary precipitate in an equal volume of 12% sulphuric acid, incubation for thirty minutes at blood heat (if cultivation is projected) and adjustment to pH 4·5. A new, smaller precipitate forms and is collected by brief centrifugation. This method of secondary concentration is complicated and its efficiency may be impaired by the presence of pus.

It will be seen that the method in common use of collecting tubercle bacilli from the urine by standing, decantation and centrifugation may vary considerably in efficiency according to whether a flocculent precipitate has formed spontaneously at the time of examination. The important researches of Hanks and Feldman and the light they have shed on the subject will undoubtedly stimulate the interest of bacteriologists in a clinical test which is often undertaken without much enthusiasm.

[1] *Proceedings of the Society for Experimental Biology and Medicine*, March, 1939.

CHEMOTHERAPY IN OTITIS MEDIA.

A report of more than usual interest on chemotherapy carried out in 396 cases of acute *otitis media* has been made by W. C. Bowers.[1] The cases in the series numbered 793. Otologists and other practitioners who have to undertake the treatment of patients with *otitis media* will find the paper worthy of careful study. It must be read critically, particularly when the author draws conclusions on a percentage basis. All the same, there are some important observations to which general attention should be drawn. The main conclusion is that chemotherapy is of decided advantage in the treatment of acute purulent *otitis media*, provided it is started early, before the condition of the mastoid has progressed to the stage of softening of the bone. When softening of the bone has occurred, the use of chemotherapy so changes the clinical picture as to make it almost impossible to determine the presence of progressive bone destruction and to ascertain the advisability of performing surgical operation. The author gives details of clinical histories which justify the statement that it may be necessary to stop administration of the drug in order to obtain a true picture. The drug cures the middle ear condition while progressive bone destruction goes on in the mastoid. Undoubtedly, then, when the clinical picture suggests the advisability of mastoidectomy, it is safer to operate. Chemotherapy has, as Bowers declares, added to the confidence of otologists; at the same time we cannot but agree with him when he adds that observations on chemotherapy must be continued "with minds open to every possibility of modification and transformation". This applies to observations other than those on the ear, and is particularly appropriate during what may be called the present sulphanilamide fashion.

[1] *The Journal of Laboratory and Clinical Medicine*, June, 1940.
[2] *The Journal of the American Medical Association*, July 20, 1940.

Abstracts from Medical Literature.

PÆDIATRICS.

Rheumatic Pericarditis with Effusion Treated with Salicylates.

ERNST P. BOAS AND MAX ELLENBERG (*The Journal of the American Medical Association*, August 3, 1940) report the successful salicylate therapy of twelve patients who were suffering from rheumatic pericarditis with effusion, and give brief summaries of the treatment of two boys aged fourteen years. Large doses of salicylates, averaging from 150 to 200 grains a day, were given for eleven days. Serial X-ray studies of the chest showed progressive diminution in the size of the cardiac shadow and resorption of the pericardial exudate. There was a sharp drop in the temperature curve. There was no demonstrable effect on the rheumatic activity *per se* as judged by the persistence of tachycardia, rapid sedimentation time of the red cells and leucocytosis. Subjectively the patients soon showed considerable improvement. In one instance Kussmaul breathing developed on the tenth day and a determination of the carbon dioxide content of the blood revealed that acidosis had developed. Salicylates were then stopped and the symptoms disappeared. The course of the disease after this followed the normal pattern of subacute rheumatism activity for weeks. The authors point out that the beneficial effect of salicylates on certain manifestations of rheumatic fever, such as arthritis and pericardial effusion, and their apparent failure to stem the progress of other lesions, such as endocarditis and myocarditis, may be related to the fact that arthritis and pericarditis represent exudative and the other lesions proliferative reactions to the rheumatic infection. Swift, after studying excised joint tissue in rheumatic fever before and after the administration of salicylates, concluded that "probably the most characteristic feature of this disease is the disappearance of exudation and the symptoms dependent upon it following the exhibition of sufficient doses of certain drugs" (that is, the salicylates). On the other hand, the proliferative response remains unaffected. The relief of the cardiac tamponade following resorption of the pericardial effusion readily accounts for the prompt subjective and objective alleviation of the symptoms. The other cardiac lesions are a result of a proliferative process and consequently are unresponsive to and unaffected by the drug.

The Treatment of Megacolon with Parasympathetic Drugs.

JOHN L. LAW (*The Journal of the American Medical Association*, June 29, 1940) records his experience in the evolution of an effective and practical method of administering acetylcholine derivatives in the treatment of megacolon in fifteen patients. Megacolon is probably caused by an imbalance of the nerve stimuli to the colon from the autonomic sympathetic or parasympathetic nervous system. There are two ætiological hypotheses: hyperactivity of the sympathetic and diminished tonus of the parasympathetic nervous system. On the basis of these hypotheses and from clinical experience there are two satisfactory methods of treatment. One is sympathectomy which blocks out sympathetic inhibitory stimuli to the colon. There are some limitations to this operative form of treatment. Children below the age of four years stand the operation poorly, with minimal benefits. In patients with considerable colonic dilatation and a thin weak musculature, operation is not apt to be successful. And, thirdly, in the absence of a dependable test for sympathetic hyperactivity, parasympathetic stimulation, rather than a block of the sympathetics, may be the best therapy in an individual case. The other method of treatment consists of the oral administration of a cholinergic drug, acetyl-β-methylcholine bromide, which stimulates parasympathetic action. No limitations in the selection of cases for treatment have as yet been encountered. The treatment with this drug and liquid petrolatum of six children suffering from megacolon was entirely successful. Two of the patients were able to discontinue the drug after three and nine months respectively. The treatment is begun by first relieving the colon of concretions and gaseous distension by the use of oil or soap-suds enemata daily for several days, and half an ounce of liquid paraffin is given each night. It usually takes from five to ten days to strike a balance with acetyl-β-methylcholine bromide, so that there are one or two stools a day. Enemata may then be discontinued, but it is best to continue liquid paraffin for several months as a mild mechanical aid. The most effective times for administration of the drug are from one-half to one hour after breakfast and in mid-afternoon. The initial average dose is 0·1 to 0·2 gramme administered after breakfast. In a few days it may be increased by giving a similar dose in the mid-afternoon. If this causes diarrhœa, the afternoon dose is omitted. When the dosage is found which produces one or two stools a day, the patient is discharged, usually taking 0·2 gramme each morning after breakfast and one to two tablespoonfuls of liquid paraffin each evening, with instructions to use an enema in the event of distension or constipation. After several months' therapy it may be possible to omit all medication except the paraffin.

Treatment of Nephrotic Œdema.

ARNOLDUS GOUDSMIT AND MELVIN E. BINGER (*The Journal of the American Medical Association*, June 29, 1940) present a regimen of treatment for patients who have a nephrotic type of œdema. It consists of rest in bed, a salt-free high-protein diet, restriction of fluids, the oral administration of potassium nitrate, and the intravenous injection of 6% solution of acacia in 0·06% sodium chloride. The authors give full particulars as to diet and dosage. They hold that the treatment is successful in 90% of the severe types of cases. Two physiological abnormalities may contribute to the appearance of œdema in the nephrotic syndrome: (i) the greatly decreased concentration of proteins of the serum and decreased colloidal osmotic pressure usually considered responsible for the deranged equilibrium between the fluid of the blood and that of the tissues, and (ii) the greatly decreased ability of the kidney to excrete sodium chloride and water. Whether or not this deficiency in renal function is conditioned in some way by the decreased colloid osmotic pressure of the serum is not known at the present time; but in the absence of another explanation the assumption seems plausible. The authors state that the scheme of treatment proposed by them appears sound from the point of view of physiological disturbance involved. The intake of water and chloride is limited. The incorporation of a rather considerable amount of protein in the diet is made with the hope of furnishing material to increase the depleted stores of the serum proteins. Potassium salts, as well as the nitrate ion, are known to cause increased excretion of chloride, and their combination in the form of potassium nitrate makes it the most potent diuretic salt available. Acacia, which is a colloidal material, in 6% solution possesses almost the same colloid osmotic pressure as normal serum. Thus the administration was recommended in the hope of correcting the greatly diminished colloid osmotic pressure of the serum of patients who have nephrosis. Determination of the colloid osmotic pressure before and after injection of acacia in these patients, however, has failed to reveal consistent differences which might be correlated with the clinical success of the treatment. The major reasons for failure of the colloid osmotic pressure to be increased after the injection of a solution of acacia is that the volume of circulating blood increases and concomitantly the constituents of the serum are diluted. There is no adequate experimental evidence that increase in volume of the circulating blood of itself provokes diuresis. On the other hand, experiments performed on healthy dogs indicate that after injection of a solution of acacia the excretion of chloride is increased. Similarly, among patients who have a nephrotic type of œdema, significant increases in the rate of excretion of chloride and water after the injection of a solution of acacia have been found. Thus, curiously enough, the administration of a substance originally recommended on the assumption that it might increase colloid osmotic pressure, appears to be therapeutically active because it facilitates the excretion of chloride and water by the kidney.

The Treatment of Pneumonia during Pertussis with Sulphapyridine.

JEROME L. KOHN, HERSCHAL J. RUBIN AND HAROLD M. HOBART (*Archives of Pediatrics*, July, 1940) have investigated the effect of sulphapyridine on the clinical course of pneumonia occurring during pertussis. Thirty-three children were treated and given the usual dosage of sulphapyridine. Of the children, 60% were under two years of age. In twelve of the children a pneumococcus was isolated from the pharynx or larynx during the illness; but sulphapyridine was given to all the patients regardless of the results of the cultures. Clinically the group in which the pneumococci were isolated did no better than those in whom other bacteria were found. There were five deaths. The authors considered that sulphapyridine had very little effect on the course of the illness in those who were severely ill. It often had an antipyretic effect out of all proportion to the clinical status. Frequently there was a recurrence of fever after the drug was discontinued. Two of the children developed agranulocytosis, and one of them died. Three other children developed leucopenia. The authors are of opinion that sulphapyridine should not be given for more

than six days and should not be repeated if there is an exacerbation of symptoms.

ORTHOPÆDIC SURGERY.

Fate of Cranial Defects Secondary to Fracture and Surgery.

M. A. GLASER AND E. S. BLAINE (*Radiology*, June, 1940) state that the healing of skull fractures depends largely on the age of the patient, the width of the fracture and its location. Linear fractures in children under six years of age disappear within six to twelve months after injury, except when the separation of a fracture has been extremely wide; in these cases the defect may persist permanently. In adults, linear fractures situated in the frontal, parietal and temporal regions begin to fade immediately; however, complete disappearance rarely occurs under seven months, and in these cases comparison with the original film shows evidence of fracture. The average time of complete disappearance of these fractures, even though fading is evident upon the X-ray film, is three to four years. Fractures in the occipital region take much longer to disappear, and in the authors' series evidence of fracture has persisted as long as eight years, though fading is apparent. In some instances the area about the fracture absorbs rather than disappears, leaving a much larger X-ray shadow defect than at the time of injury. This may occur in children as well as in adults. The result is a permanent defect. In depressed fractures without elevation the fragments become rounded and unite, and the lines of fracture cannot be detected, though the depression is apparent. In operative defects in which the bone has been removed, or in cases of depressed fracture in which the fragments have been removed, the cranial defect never becomes smaller, the only change being a rounding of the edges.

Tuberculosis of the Mandible.

C. M. MENG (*The Journal of Bone and Joint Surgery*, January, 1940) states that tuberculosis of the mandible, although heretofore considered to be rare, has been observed not infrequently. Of the fourteen cases reported by the author, eight were seen within a period of two and a half years. The majority of the patients belonged to the second and third decades of life. Swelling, discharging sinuses, and occasionally trismus of the jaw were the chief complaints. About 43% of the patients in this series had tuberculous lesions in other bones of the body, and tuberculosis of the skull was coexistent in 29%. In ten of the cases in which the chest was studied radiographically, evidence of tuberculosis either in the lungs (eight cases) or in the pleura (two cases) was seen. It appears that the tuberculous infection of the mandible is almost always hæmatogenous, originating from a primary focus elsewhere in the body, most likely in the lungs, although the mandible may be involved from extension of a tuberculous lesion of the mucous membrane of the oral cavity or from infected gingivæ about carious teeth. In rational therapy general antituberculous measures must be emphasized in addition to local treatment of the tuberculous lesion of the mandible. Radiographically, either very slight change or extensive involvement of the mandible may be seen. Rarefaction and necrosis of the bone are evident; involucrum is scanty and often absent altogether; sequestration, however, is frequently observed. Small sequestra, although often not detectable in the skiagrams, may be found in a bone cavity at operation. The entire length of the ramus may be sequestrated or even displaced far from its original site. Pathological fractures are common.

Fractures of the Neck of the Femur.

F. E. GODOY-MOREIRA (*The Journal of Bone and Joint Surgery*, July, 1940) describes a device and technique that he has used successfully in ten cases of fracture of the femoral neck. He has designed a special stud-bolt screw, with its centre cannulated to permit the passage of a Kirschner wire. The instrument consists of a screw proper, a cupped flange, with an inclination to suit the bone surface, and a locking nut, which fits into the hollow flange. A new set of instruments for insertion of the screw was also devised by the author. The first step in the operation is to obtain correct reduction of the fracture. A Kirschner wire is introduced through the centre of the neck in the usual manner. A channel is now made for the screw by means of an electric burr, grooved for the purpose of fitting over the wire. The burr perforates the bone for two centimetres, which is sufficient to receive the cupped flange. With a longer and narrower drill the channel is extended to the required length. The screw is inserted along the wire with a special wrench, followed by the flange and locking nut. The latter is manipulated until its thread catches the screw. By tightening the nut the screw becomes tight, and so draws the fractured ends together. No plaster cast is required, and the patients are allowed to walk in from two to four weeks. This method does away with the need of a mallet required in introducing the Smith-Petersen nail, and also obtains firmer impaction. A second operation is not required, as may become necessary when a nail becomes loose.

Fracture of the Shaft of the Femur in Children.

A. B. LE MESURIER (*The American Journal of Surgery*, July, 1940) details the treatment of fractured shaft of the femur, applied in the Hospital for Sick Children, Toronto. The majority of the patients are over two years of age, and the commonest site of fracture is in the middle third of the bone. Practically all are treated on a Thomas splint, with the patient lying on an inclined Bradford frame. Strapping is applied to the limb in the usual manner, and the splint is applied without anæsthetic. The end of the splint is tied to the top rail of the lower end of the bed. The Bradford frame is inclined at first about one in three, that is, the lower end of a six-foot frame is raised two feet higher than the head end. Extension is thus obtained by the patient's own weight, and adjustment of the tilt will alter the amount of extension. The splint is not supported in any way. Sufficient movement of the child is readily obtained for feeding and nursing purposes, and the patients tolerate the position well. Every child admitted to hospital with suspected fracture is immediately treated along these lines, even before an X-ray examination is made. The next step is to obtain accurate reduction, the standard aimed at requiring full-length reduction, parallel general alignment, contact of the fracture surfaces, the absence of obvious *varus* or *valgus* deformity, and no *genu recurvatum*. Fairly strong traction on the frame often effects satisfactory reduction in two or three days. Backward displacement can be corrected by tightening the sling under the fracture, and lateral displacement improved by pulling the fragments towards either bar of the splint. Occasionally manipulation under an anæsthetic is necessary. After four weeks union is moderately firm and a plaster spica is applied for a further six weeks. The spica is then removed and the child is allowed to kick about in the bed for a week before walking is commenced. With small infants the Bryant method of vertical suspension of both legs is adopted with excellent functional results, although the position obtained is not always perfect.

Degenerative Arthritis of the Knee Joint.

G. E. HAGGART (*The Journal of Bone and Joint Surgery*, July, 1940) discusses the surgical treatment of degenerative (hypertrophic) arthritis of the knee joint. He considers that conservative treatment, based on the principles of rest and physical therapy, is unsatisfactory. The studies of Keefer, Parker, Myers and Irwin of 100 knee joints show that over the age of thirty most knee joints reveal gross anatomical changes. The two regions most markedly affected by the degenerative changes are the patella and the patellar surface of the femur. The author believes, therefore, that removal of the patella is a reasonable procedure to relieve symptoms and to improve function. This operation was performed on twenty patients. Exercises before and after operation were instituted, and the cooperation of the patients was essential, particularly in obtaining active movements after operation. The parapatellar incision was used. In addition to the patella, hyperplastic synovial membrane was excised, with the exception of the posterior compartment, together with exostoses, and one or both menisci when damaged. A short period of immobilization was followed by active exercises. Improvement was shown by nineteen of the patients. The one failure occurred in a patient with mixed rheumatoid and degenerative arthritis.

Herniation of Intervertebral Disk.

HERMAN F. JOHNSON (*The Journal of Bone and Joint Surgery*, July, 1940) analyses forty cases of low back pain associated with sciatic radiation. He states that when intractable sciatic pain persists in spite of conservative measures, such as traction, immobilization and physical therapy, the treatment is operative. Twenty-four patients were operated on, and hemilaminectomy gave adequate exposure in all but two cases. In one case, in which the final diagnosis was arachnoiditis, an extensive laminectomy was performed, and this was followed by spinal fusion. The average age of the patients was forty-one years. A history of trauma was present in 62·5% of cases, and the average duration of symptoms was five years. Positive neurological findings, such as diminished or absent ankle jerk and impaired cutaneous sensation, were present in 79%. The protein content of the cerebro-spinal fluid was above normal in 65%, and lipiodol studies showed filling defects in twenty-two of the twenty-four patients operated on. Complete relief was obtained in 83%, and in 4% there was no improvement of symptoms.

Special Articles on Psychiatry in General Practice.

(Contributed by request.)

XV.

STATES OF MENTAL DEFECT: OLIGOPHRENIA: CONGENITAL MENTAL DEFICIENCY.

THE term mental defect or mental deficiency is one that is often very loosely used to describe almost any form of psychosis. This can be understood, for a mental disorder is in fact an indication of some defect in the mental mechanisms. If the more accurately descriptive term of intellectual defect were used, the meaning would be clear. Mental deficiency therefore denotes intellectual defect arising from birth or a very early age. In such cases an adequate or independent social adjustment is impossible. Many different names have been applied to this condition, to the extreme confusion of those who are trying to obtain some conception of its meaning. These terms are feeble-mindedness, oligophrenia, hypophrenia and amentia. The last is particularly misleading, as it is also in common use on the Continent to describe an organic syndrome with confusion and disorientation. It is better to avoid its use altogether.

The diagnosis of mental defect may be quite simple in the case of low grades of mentality; but in the higher grades it may be necessary to make use of certain tests to obtain an accurate estimate of the degree of deficiency present. It may be said, generally, that the diagnosis is more easily made the older the child is. A very young child, because of its failure of cooperation, may present considerable difficulty when it is tested, while fluctuations of attention and indifference to the procedure may give rise to the conclusion that the child is retarded when such is not the case. In gross cases, when deformities of the skull and various so-called stigmata of degeneration are present, there can be no possible doubt about the nature of the condition. To carry out tests in such a case is merely to be redundant. Before a brief description of these tests is given and their value in determining the degree of defect is explained it might be well to mention the accepted classification of mental defectives.

The Three Main Groups of Mental Defectives.

For practical purposes mental defectives may be divided into three groups, those comprising the idiot, the imbecile and the feeble-minded. The term moron is applied to the last group in America. These groups have been clearly defined by the *Mental Deficiency Act* in England and by the *Lunacy Act* of New South Wales as follows:

Idiot: "A person so deeply defective in mind from birth or an early age as to be unable to guard themselves against common physical dangers." This is the lowest form of mental development, and one which is readily recognizable from an early age. Marked deformities are usually found and are often associated with epilepsy and various paralyses. If an idiot can speak at all, he utters only a few, often quite unintelligible, words, and he is incapable of feeding himself in a normal fashion or of looking after himself. He is prone to disease and requires constant care and attention in an institution if life is to be prolonged, although there appear to be very few reasons why it should be. The *Lunacy Act* of New South Wales provides only for the certification of idiots; the other groups are not mentioned, and when it is necessary for persons in those groups to be certified they have to be described as insane, which does not seem to be exactly correct.

Imbecile: "A person in whose case there exists from birth or from an early age mental defectiveness not amounting to idiocy, yet so pronounced that they are incapable of managing themselves or their affairs, or, in the case of children, of being taught to do so." Many imbeciles are not fit to be outside an institution because of their childishness, occasional outbursts of temper and tendencies in adolescence to sexual offences, of which they have no true appreciation. They are certified on the grounds of their intellectual defect, although the act makes no provision for this. They cannot earn a living and readily become the tools of the criminally minded or the butts of the misplaced humour of the thoughtless. They are often stolid ungainly people, with poorly coordinated muscular hovements. They can be educated to a certain extent and may be capable of performing simple tasks. The quiet and amenable types may never reach an institution and, as they are frequently very affectionate, their parents may make every effort to avoid this, particularly in the case of an only child. Mongols, to which reference will be made later, make up a large proportion of this group. In comparison with young children of the same mental age, imbeciles are deficient in a capacity for original adaptation, natural vivacity and spontaneous inquisitiveness.

Feeble-Minded or Moron: "A person in whose case there exists from birth or an early age mental defectiveness not amounting to imbecility, yet so pronounced that they require care, supervision and control for their own protection or the protection of others." From this group are recruited many criminals. The figures given vary from 20% to 69% of all offenders. Feeble-minded persons present one of our greatest sociological problems. They lack the capacity for sustained effort, drift from one unskilled job to another, and may easily gravitate to a life of crime. It is only in rare instances, under our present lunacy laws, that they can be sent to a mental hospital for supervision and control; but a definite step forward has recently been made in our dealing with the criminal moron, and reference to this will be made later. This group is nearly four times as great in the general population as the other two combined. In this group also belong the *"idiots savants"*, who, in striking contrast to their backwardness in other directions, show a special aptitude in a certain direction, such as music, painting or in remembering dates and figures.

The Estimation of Mental Deficiency.

The method used for estimation of the degree of mental deficiency is the Stanford revision of the Binet-Simon method. The latter was in use in France and it was revised and modified to suit conditions in America and is now in universal use for this purpose. It consists of a series of carefully graded tests, the successful performance of which does not depend on ordinary schooling; they can therefore be applied to those who have been unable to make regular attendances for a variety of reasons. They commence with such simple tests as asking the child to point to certain parts of the body and increase in complexity until quite complicated problems are presented. It has been accurately determined what a child at each age from three to sixteen should be capable of performing, and the mental age, as opposed to the chronological age, can thus be obtained from the results of these tests. The mental age, therefore, is that represented by the number of tests successfully carried out by the child, and in the case of a normal child will be approximately the same as the actual age. The result is expressed as a quotient by dividing the mental age by the actual age and multiplying by one hundred. This is known as the intelligence quotient or, more simply, the I.Q. For example, if a child of twelve passes all the tests up to and including those of the eighth year, but no more, its mental age is eight years and its intelligence quotient $8 \div 12 \times 100$, which equals 66. For the purpose of the tests the adult mental age is accepted as sixteen. Similar results are achieved by the Porteous maze test, which is self-explanatory, and the Koh's block test, in which a series of coloured blocks are arranged to form given designs of increasing difficulty, according to the mental age of the subject. These are of greater value for illiterates, but the Binet-Simon method remains the most commonly used and the most satisfactory for general purposes.

The accepted intelligence quotients for the three grades of mental defectives are: idiot, below 20 to 25; imbecile, 25 to 50; and feeble-minded, 50 to 70. In the case quoted above the child would be rated as feeble-minded. Various other types are also described in the classification, but these are rather of academic interest than of practical value. They include: genius, above 140; very superior intelligence, 120 to 140; superior intelligence, 110 to 120; normal or average intelligence, 90 to 110; dulness, 80 to 90; and borderline deficiency, 70 to 80. The higher figures are obtained when the subject succeeds in tests beyond the actual age or, in the case of an adult, answers all the questions in year XVIII or "superior adult".

Apart from the intelligence tests there are other signs which indicate the presence of mental defect. The most striking of these are the "stigmata of degeneration". These include abnormalities of the size or shape of the skull, prognathism, deformities of the pinna of the ear, narrow and highly arched palate, mongolian cast of countenance, and malformations of the limbs and fingers. Restlessness, tantrums, wandering from home, acts of cruelty towards younger children and animals, outbursts of screaming, resistiveness, lack of attention and interest, inability to learn the simplest of lessons, and backwardness in walking and talking are all indications of mental retardation.

Other Clinical Types and Causes of Mental Defect.

In addition to the three grades of defect there are certain clinical types which are of interest. The most striking of

these is the mongol, who comprises about 5% of defectives of all ages. He has a characteristic Asiatic appearance, with a small rounded brachycephalic skull, a large fissured tongue, a short broad nose with a depressed bridge, stumpy fingers, and narrow, oblique and slit-like palpebral fissures. It is difficult to believe that a group of such children, seen together, are not related. The cause of this condition has exercised the speculative fancies of psychiatrists for many generations, and many and varied are the theories offered to explain it. It has been suggested that it is a form of atavism; the endocrines have, of course, been blamed; and alcoholism, consanguinity, tuberculosis and syphilis in the parents have been investigated as possible factors and discarded. Mongolism seems to occur more frequently in the children of older mothers; but even this has not been satisfactorily established. This type is usually very good natured, affectionate and easily amused. Mongols are generally imbeciles.

Microcephaly is another clinical variety. Here we have a cone-like oxycephalic skull with a circumference of less than seventeen inches, a receding forehead and chin, and a prominent nose, giving the child a peculiar bird-like appearance. In spite of the relatively small brain, microcephalic children are not of a very low order of intelligence, and many manage to earn some sort of a living because of their essential industry.

Cretinism is a comparatively rare form of defect in this country. It is due to a deficiency in thyreoid secretion. It is customary to divide children suffering from this condition into three groups: the cretins, who lack reproductive and intellectual powers and are mute; the semi-cretins, who have reproductive powers and some rudiments of language; and the cretinoids, who can be educated to a certain degree, but who never reach the average in intelligence. The characteristic features are stunted growth, protruding tongue, dry skin, scanty hair, short limbs and clumsy gait. The response to thyreoid medication in early life is most striking and gratifying. Rapid improvement, both mental and physical, is common. This serves to explain the frequent use of thyreoid in all cases of mental deficiency, although in other forms the optimism is quite unjustified.

Hydrocephalus is a source of mental defect, as might be expected from the distortion of the cerebral tissue and the consequent failure of function. The degree ranges from idiocy to feeble-mindedness. The physical outlook, fortunately, is poor, few hydrocephalic children reaching adult life.

Other clinical groups include: the scleroses, in which there is proliferation of neuroglia, diffusely or in nodules, the latter often being associated with *adenoma sebaceum* and epilepsy, when it is known as epiloia; the paralytic form, which commonly results from hæmorrhage during prolonged labour; the syphilitic and epileptic types; and the inflammatory variety, which arises from inflammation of the brain and meninges due to one of the infectious fevers. In all these there is usually a gross degree of mental deficiency.

A clinical variety worthy of note is amaurotic family idiocy. This, as its name implies, is a familial condition. It is most commonly found among Jews and manifests itself in idiocy, spastic or atonic paralysis of the limbs and blindness due to optic atrophy. It usually appears after the fourth month and reaches a fatal termination before the age of two. A similar condition may develop later in life and also be associated with paralysis and blindness. The distinguishing feature in the former is the appearance of a cherry red spot at the macula. Quite recently a hitherto unrecognized type of familial mental deficiency has been described. This is associated with the excretion of phenylpyruvic acid in the urine. It is a unique instance of developmental deficiency, biochemical in character. Extrapyramidal manifestations are also present. The posture is generally that of flexion with rigidity of flexor and extensor muscles and tremors and athetosis.

Educational retardation as opposed to actual mental defect may sometimes occur, and shows itself as a failure of capacity to assimilate ordinary subjects taught at school, particularly mathematics. Defects of vision or hearing may play a part, and the very scrappy examinations made of school children by visiting doctors many years ago may well have failed to elicit these failings and may have resulted in children being dubbed backward without any real justification. Even those who put up a poor performance at school may prove themselves in after-life because of a certain practical ability and fund of common sense which are not by any means common accompaniments of scholastic distinction. There are cases, however, in which an absolute dullard, who is obviously backward at school, ultimately seems to become more alert mentally and succeeds in making a satisfactory adjustment to life and the demands of society. Such a case was that of a girl who appeared to be the worst type of delinquent and of a very poor order of intelligence. She was in and out of homes of correction repeatedly, but eventually married and to all intents and purposes became a normal happy housewife, greatly to the surprise of all who had come in contact with her.

The Treatment of Mental Defectives.

We now come to the problem of how we are to deal with these defectives which have been described. Some can be managed at home, as they are tractable and affectionate and their parents prefer this. Others require segregation in an institution or a colony for their own protection or that of others. It must be remembered that very often they have the sexual development of an adult without the adult's capacity for control. Female defectives are only too often the prey of unscrupulous males. The tendency of many to drift into crime or become the catspaws of criminals has already been mentioned. Unfortunately, this most often occurs in the case of the higher grades of mental deficiency, when it is difficult or almost impossible to deal with them under the terms of the *Lunacy Act*. The recently passed *Criminal Mental Defectives Act* of New South Wales will go a long way towards solving the problem in this State. Briefly this empowers the authorities to bring any criminal defective who has served a sentence of more than two years for a previous offence before a board, consisting of two doctors and a magistrate, who may then order him to be detained for an indeterminate period in an institution set apart for the purpose.

In the case of the idiot and the low-grade imbecile little can be expected from training, and the most that can be done is to look after their physical welfare. In the higher grades something may be done to make those affected self-supporting at least. Farm work and simple trades may be taught, with the hope of achieving some useful purpose. During childhood efforts should be made to teach the child to dress and to look after himself and to keep himself clean. He may learn to avoid common dangers. It is desirable that these children should be removed from the home if there are other children, as the effect on the other children may be undesirable and also they may, with childish thoughtlessness, be cruel and unsympathetic. In addition, with institutional care of the defective the family is usually able to make a better social and economic adjustment.

From the community point of view the low-grade defectives do not constitute an important problem. Their number is small compared with the higher grade, and many die at an early age. They do not constitute a eugenic problem, since the defect is rarely of familial origin and practically none ever procreate. With the higher grades the vexed question of sterilization arises. The very mention of this generally gives rise to a howl of execration from self-appointed guardians of civil liberties. It is a highly controversial subject which need not be discussed here, except to point out that it has certain economic advantages in reducing the numbers which require institutional care and the consequent expense to the State; but these may be offset by the spread of venereal disease in those who regard it as giving them sexual licence, and by the fact that this procedure may add to *libido* and thus increase the incidence of assaults on young children.

Nature herself solves the problem of the mental defective for us by terminating life very early in the worst cases; we can do our share by urging early institutional care and training of the higher grade defectives and by endeavouring to encourage them to make some sort of social adjustment, so that economically, socially and intellectually they may not be too great a burden on an already overtaxed community.

JOHN MCGEORGE, M.B., Ch.M., D.P.M.,
Honorary Physician, Psychiatry Clinic, Royal Prince Alfred Hospital.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on May 15, 1940, at the Royal Melbourne Hospital. The meeting took the form of a number of clinical demonstrations by members of the honorary staff of the hospital. Parts of this report appeared in the issues of August 10, August 24, September 21, September 28 and October 5, 1940.

Hodgkin's Disease.

DR. R. KAYE SCOTT showed a series of patients suffering from Hodgkin's disease. He drew attention to the results of Watt (*The British Medical Journal*, October 10, 1936, page 712), who showed that approximately 20% of patients

died within the first two years, that 55% died within the two-year to five-year period, and that approximately 25% survived for from five to twelve years.

The pathologist would rarely give an unequivocal opinion on a suspected case of lymphadenoma. Few clinicians were prepared to believe that such patients could survive for more than five years, and if survival occurred the diagnosis was immediately questioned. Three patients were shown who had survived for nine, twelve and fourteen years. In two of these cases the pathologists could not decide between Hodgkin's disease and reticular-cell sarcoma; in the third case the diagnosis lay between Hodgkin's disease and lymphosarcoma.

Patients with Hodgkin's disease divided themselves sharply into two groups in their response to treatment by radiotherapy. Some showed recession of the glandular enlargement, which remained inactive after the usual two or three routine courses of therapy had been given. The patients needed no further treatment during the succeeding years, and in this group real cures might be expected. The second and more numerous group showed temporary or incomplete response, and continued courses of therapy were needed either to treat new manifestations of the trouble or to inactivate recrudescences. But by close and regular observation of patients in this latter group, many of them could be kept going for a period of years, provided immediate treatment was administered on the appearance of each indication. One of the patients shown had first noticed glandular enlargement twelve years previously and had received continual treatment since. In the previous week she had presented a fresh glandular involvement.

Patients with generalized glandular enlargement and pyrexia were difficult problems on account of their limited tolerance. It was rarely possible to give to the glands a dosage up to levels sufficient to cause immediate complete resolution; but by limitation of the size of the fields and by the administration of a small daily dosage to each area in turn, the temperature was often found to subside. The total amount of therapy, however, was limited by the resultant leucopenia. Neither the Pel-Ebstein type of Hodgkin's disease nor the type associated with continual pyrexia had been satisfactorily explained.

In hospital practice it was much more common to find advanced cases with massive generalized glandular enlargements, and these patients in general did poorly.

Prognosis was much better when only one or two series of glands were involved. Hodgkin's disease was seen quite frequently in children, and the same rule applied: in generalized cases the response was bad, while patients with restricted areas of involvement had a reasonable chance. In children the granulomatous process readily attacked the osseous system, the most frequent manifestation being periostitis, particularly of the tibia, which caused local pain, swelling and tiredness. In the X-ray film the periosteum was seen to be lifted off the subjacent bone, the latter showing some rarefaction in gross cases. The process disappeared very rapidly following one or two doses of X-ray therapy. Films were shown illustrating this condition.

Dr. Kaye Scott showed a child, aged thirteen years, suffering from advanced generalized (proved) Hodgkin's disease. Ten days previously she had complained of a tender swelling among her hair. Examination revealed a flat, raised, hard swelling arising from bone immediately lateral to the lambda; the scalp moved over its surface. In the ten days it had subsided by half after radiotherapy. X-ray films of the area were inconclusive. It was regarded as a focus of Hodgkin's tissue arising under the periosteum, a rare manifestation in this situation.

Dr. Kaye Scott went on to say that bone involvement in adults was common in advanced stages, and here lymphadenomatous tissue might cause destruction in any bone containing red marrow, the vertebræ being the most common site. He showed a film illustrating destruction in several thoracic vertebræ and ribs.

Dr. Kaye Scott then showed several patients to illustrate his remarks.

A female patient, aged forty-seven years, had been first seen by Dr. Kaye Scott in August, 1926, after resection of upper deep cervical lymph glands by Dr. H. R. Dew. The glands had been swollen for five weeks. Examination of sections revealed "either reticular cell sarcoma or Hodgkin's disease". The patient received deep X-ray therapy between 1926 and 1928. Enlarged axillary glands appeared during this time, but regressed. The patient had had no treatment since 1928 and remained well.

A male patient, aged fifty-nine years, was seen in February, 1931, when he had enlarged glands in each side of the neck and in the left groin. Examination of sections suggested Hodgkin's disease, but the periphery was more active; this suggested sarcomatous change. At the same time the patient had a parotid tumour treated with radium needles. It completely disappeared. Deep X-ray therapy to the glands had been last given in 1933, and the gland fields remained clear. Recently the patient had contracted low-grade pulmonary tuberculosis.

Another patient, a female, had first noticed enlarged glands in the left side of her neck in 1928; these were excised two years later and again a year later after a recurrence. Examination of sections by Professor MacCallum revealed "either Hodgkin's disease or lymphosarcoma". In 1932 the left side of the neck was treated for a further recurrence. The swelling of the glands subsided, but a residual swelling was treated with radium needles, as a diagnosis of sarcoma had been suggested. In 1937 enlargement of the spleen and of the mesenteric and epigastric glands was noted; and this enlargement had subsided with treatment. The patient reported ten days before the meeting with an enlarged right preauricular gland and several enlarged glands in the right side of the neck. Dr. Kaye Scott said that these new foci were receiving treatment now, twelve years after the onset of her disease.

A boy, aged eleven years, was sent from the Children's Hospital in April, 1934, with a mass of glands six to eight centimetres in diameter in the right side of the neck. Enlargement of the glands in the anterior and posterior mediastinal spaces was demonstrated. The gland masses subsided and the mediastinum now appeared quite normal. The boy was well, six years after the onset. Small slightly fibrotic glands were palpable in the right side of the neck.

A male patient, aged fifty-three years, had reported recently with massive glandular enlargement in the left side of the neck and smaller enlarged glands in the right side of the neck, the axillæ and the groins. The enlargement had practically disappeared after two courses of deep X-ray therapy. Dr. Kaye Scott said that clinically the disease fitted in with a lymphadenomatous process until an enlargement of the right lachrymal gland occurred; this "melted away" after one or two doses of irradiation. This lachrymal gland enlargement, as confirmatory evidence, justified the pathologist in refusing to diagnose Hodgkin's disease; it also called to mind the condition of Mikulicz's disease, which was now being classed among the reticulo-endothelial neoplasias (Paterson and Tod, *American Journal of Roentgenology and Radium Therapy*, Volume XLII, 1939, page 820). It was possible that this case illustrated a diffuse reticulo-endothelial hyperplasia involving lymphatic and lachrymal glands.

The Results of Perurethral Resection of the Prostate.

DR. HAROLD MOORE showed a series of patients who had been subjected to perurethral resection during the last five years. In none of these patients was the prostate of the very large soft type. All of them derived great subjective benefit from the operation, though two of them still had a quantity of residual urine varying between two and three ounces. These two patients were quite satisfied with their condition and were unwilling to undergo any further operation.

One patient was of particular interest, in that he had a small adenomatous nodule within the posterior portion of the urethra, and removal of this nodule without any further measures resulted in immediate loss of his pain and in freedom from all symptoms. Dr. Moore said that this particular patient could not have been dealt with satisfactorily in any other way.

A point illustrated was the importance of operating in the presence of severe symptoms if cystoscopic examination revealed a grossly trabeculated bladder. This trabeculation was an indication of obstruction, even if the prostate was not grossly enlarged and if there was no residual urine. One such patient, who had a small prostatic bar which had been resected, claimed complete relief from pain and nocturnal frequency of micturition. Another patient had been under observation for some years, suffering from a chronic urinary infection. He suffered from intermittent hæmaturia, rigors and occasionally complete retention of urine. X-ray films taken after the intravenous injection of "Uroselectan" revealed tremendous dilatation of both ureters, with poor renal function. There was no enlargement of the prostate; but the bladder was trabeculated and there was a large amount of residual urine. Resection of the posterior margin of the prostate gave great relief. The amount of residual urine was reduced to three ounces.

Dr. Moore said that at the Royal Melbourne Hospital it had been the practice to limit resection to small prostates with a definite bar or prostates with an enlargement of the middle lobe alone. Occasionally resection was carried out when a patient had enlargement of one lateral lobe; but if both lateral lobes were enlarged it was thought preferable to perform a suprapubic enucleation. The reason for this was that, no matter how much was resected, there

was always the tendency for the remainder of the gland to push the lobes together so that the obstruction was continued.

Tuberculous Ureter.

Dr. Moore then showed X-ray films of the stump of a tuberculous ureter which was causing the continuation of symptoms after nephrectomy for a tuberculous kidney. The specimen removed at operation was also shown. Since the second operation the patient's symptoms had already been ameliorated. Dr. Moore said that this patient illustrated the usual procedure adopted at the Royal Melbourne Hospital in nephrectomy for tuberculosis. At the primary operation the kidney and as much of the ureter as could be easily reached were removed through the usual loin incision. As a rule there was no further trouble from the stump of the ureter; but every now and then a patient did have trouble, as in the case demonstrated. In these circumstances it was held that a second operation some months later for removal of the remainder of the ureter was the proper treatment and did not impose such a strain on the patient as an attempt to deal with the whole of the ureter at the first operation.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 199, of September 26, 1940.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Volunteer Reserve.

Appointment.—Robert Samuel Irwin is appointed Surgeon Lieutenant (on probation), dated 1st September, 1940.

AUSTRALIAN IMPERIAL FORCE.

Australian Army Medical Corps.

To be Majors.—Captain T. G. Swinburne, J. B. Somerset, E. E. Dunlop, 1st May, 1940; and T. U. Ley, 1st June, 1940.

The regimental seniority of Officers of the Australian Army Medical Corps in the Australian Imperial Force is as follows: Colonels S. R. Burston, C.B.E., D.S.O., V.D., N. L. Speirs, V.D., W. W. S. Johnston, D.S.O., M.C., E.D., H. C. Disher, J. Steigrad, Sir T. P. Dunhill, K.C.V.O., C.M.G., W. E. Kay, D.S.O., V.D., A. P. Derham, M.C., F. K. Norris, R. A. Money, M.C., E.D., W. E. Summons, O.B.E., V.D., N. H. Fairley, O.B.E.; Lieutenant-Colonels K. B. Fraser, K. J. G. Wilson, H. G. Furnell, J. K. Adey, O.B.E., F. H. Beare, L. E. LeSouef, J. C. Belisario, E. M. Sheppard, A. H. Green, L. G. Male, A. J. Cunningham, L. C. E. Lindon, E. B. Jones, A. S. Walker, D. W. McCredie, M.C., H. F. Summons, J. G. Hayden, E. L. Cooper, A. W. Morrow, S. H. Lovell, B. S. Hanson, D. B. Loudon, C. W. B. Littlejohn, M.C., A. C. Thomas, J. R. M. Beith, D.S.O., V. M. Coppleson, C. G. McDonald, F. J. Clark; Major (Honorary Lieutenant-Colonel) H. C. Nott; Majors A. H. Powell, D.S.O., R. M. W. Webster, M.C., M. L. D. McKeon, H. McLorinan, W. Park, G. B. G. Maitland, D.C.M., N. B. White, G. C. Burston, T. A. Parry, D. M. Salter, G. G. L. Stening, R. J. Stabback, N. Eadie, J. Gillespie, R. J. A. Henderson, R. H. Russell, K. W. Starr, N. H. W. Saxby, N. W. Francis, A. L. Dawkins, K. S. Richardson, J. B. Colquhoun, J. M. Dwyer, J. H. Stubbe, H. R. Love, H. M. Fisher, W. W. Lempriere, E. Bailhache, H. M. Trethowan, M. A. Rees, F. R. Hone, I. J. Wood, G. F. Hill, T. Y. Nelson, K. C. Ross, L. F. Dods, A. O. Davy, I. M. Mackerras, E. V. Keogh, J. O. Smith, E. S. J. King, R. V. Graham, W. E. A. Hughes-Jones, A. R. Colwell, S. Plowman, R. N. Howard, N. L. Sherwood, T. G. Swinburne, J. B. Somerset, E. C. Palmer, C. A. M. Renou, O. E. Nothling, R. B. Maynard, W. E. Langford, F. K. Mugford, A. D. Byrne, R. T. Binns, J. S. Crakanthorp, E. E. Dunlop, S. W. Williams, F. W. Niesche, J. C. G. White, R. McK. Rome, C. W. Nye, A. D. Matheson, D. A. Cameron, F. N. Chenhall, J. D. Palandri, T. U. Ley, A. F. Hobson, F. K. Wallace, W. P. MacCallum, O. E. J. Murphy, A. W. L. Row, H. M. Cutler, A. J. Murray, J. H. Halliday, K. B. Noad, E. F. Thomson, R. H. B. Bettington, C. S. Colvin, D. J. Thomas, D. O. Brown, E. Ford, H. Rayson, E. F. West, J. B. Hamilton, C. H. Osborn, G. B. D. Hall, R. Jeremy, S. V. Marshall, W. L. MacDonald, K. B. Voss, M. P. Susman, G. N. Lorimer, M.C.; Captains J. E. Sewell, J. F. Akeroyd, S. W. Bryan, C. W. K. Hardy, J. F. J. Cade, A. L. Carrodus, J. Kingsley, G. N. Young, J. S. Chalmers, L. G. Hill, T. L. Tyrer, J. M. Blair, I. H. Sender, C. R. Blomfield, C. F. Marks, F. D. Stephens, R. J. Wheeler, R. S. Smibert, S. L. Seymour, A. W. Robertson, J. S. Peters, C. H. Johnston, J. Ray, R. J. Humphery, A. M. Macintosh, H. R. Smith, L. E. Rothstadt, R. H. Macdonald, P. C. Thomas, A. K. Green, R. Warden, W. D. Refshauge, E. P. Cherry, W. M. Irwin, P. A. Tomlinson, C. H. Selby, W. K. Myers, I. F. Vickery, A. F. Janes, V. G. Bulteau, R. B. M. Pilcher, E. L. Davis, A. R. Hazelton, R. J. Hoy, D. W. Brummitt, D. G. Perrett, R. G. Champion de Crespigny, J. R. Magarey, A. A. Moon, J. L. Holme, A. M. Johnson, J. A. F. Flashman, W. W. Gunther, E. J. C. Molesworth, S. Crawcour, B. L. Hellings, I. T. Cameron, J. R. Goding, J. B. Devine, J. J. Searby, D. R. C. Wilson, N. J. Bonnin, H. F. G. McDonald, S. J. Douglas, R. F. West, T. H. Ackland, B. Moore, P. Braithwaite, J. R. Radcliff, A. D. A. Mayes, R. R. Winton, R. E. Maffey, J. O. Lavarack, M. S. Alexander, R. S. Wilkinson, A. L. Johnston, H. W. Anderson, A. E. Vivian, G. T. Gibson, R. R. Anderson, J. J. Ryan, R. R. Andrew, C. Gurner, R. W. Johnson, G. W. Pottinger, K. H. Heard, N. H. Robinson, J. B. W. Meredith, R. Drummond, R. A. Playoust, K. B. Armstrong, P. H. Macindoe, E. A. Hedberg, F. D. M. Williams, J. F. McCulloch, K. J. J. Dorney, E. F. MacKenzie, W. J. L. Duncan, E. R. Crisp, R. Officer, N. R. Godby, N. P. Wilson, S. I. Weir, G. Kaye, R. F. A. Becke, R. F. Matthews, L. V. Armati, H. O. G. Selle, R. G. B. Cameron, J. I. Loewenthal, W. S. L. Stening, W. M. Ada, L. G. Travers, E. L. Corlette, E. V. Barling, D. M. Yeates, K. B. Burnside, G. C. H. Hogg, N. F. Freemantle, A. G. G. Carter, R. B. Perrins, E. P. Row, W. P. Wippell, C. B. Cox, J. F. Sullivan, C. H. W. Lawes, W. Freeborn, M.M., A. V. Jackson, W. D. Sturrock, R. Motteram, P. C. R. Goode, F. J. Fenner, Max Mayrhofer, D. W. MacPherson, B. St. P. Gillett, A. J. King, A. G. C. Budge, Z. Schwartz, J. C. Stewart, D. G. Duffy, H. D. Steward, J. V. Latham, S. J. M. Goulston, W. H. Long, A. H. McGregor, F. E. Gallash, A. B. Sullivan, C. R. B. Blackburn, D. N. F. Leake, W. G. Holt, I. A. Brodziak, L. S. Loewenthal, E. H. Goulston, D. G. Croll, D. B. Wherrett, L. G. Morton, K. F. Russell, J. E. Clarke, N. R. Wyndham, H. B. Woolford, N. H. Rose, P. I. A. Hendry, A. K. Barrett, S. E. J. Robertson, M. T. Cockburn, L. W. Johnston, G. V. Mutton, J. K. Harbison, B. M. Carruthers, B. K. Rank, G. V. Rudd, A. L. B. Webb, J. P. Horan, A. J. M. Sinclair, J. W. McNamara, C. D. Jermyn, A. K. Lendon, L. A. Atkins, J. C. English, W. Aitken, T. H. Steel, P. J. Parsons, H. I. Turnbull, K. S. Day, G. V. Smith, J. F. Park, M. R. Gold, D. G. Picone, I. L. Duncan, R. L. Cahill, A. J. Kennedy, P. G. Hayes, R. L. Jeffrey, A. Fryberg, R. B. Speirs, G. C. Moss, D. F. Mercer, E. W. Levings, B. H. Anderson, J. F. C. C. Cobley, H. M. De Burgh, E. B. Jones, G. C. Halliday, H. S. Stephens, H. O. Lancaster, A. P. Findlay, R. M. Mills and R. H. Formby.

The notification respecting the seniority of officers of the Australian Army Medical Corps in the Australian Imperial Force which appeared in Executive Minute No. 3/1940, promulgated in *Commonwealth Gazette*, No. 4, of 11th January, 1940, is cancelled.

To be Lieutenant-Colonels—Major (Temporary Colonel) E. L. Cooper, Australian Army Medical Corps, 3rd Military District, 16th May, 1940; Major (Temporary Lieutenant-Colonel) A. W. Morrow, Australian Army Medical Corps, 2nd Military District, 22nd May, 1940; Lieutenant C. W. B. Littlejohn, Reserve of Officers, 3rd Military District, 20th May, 1940; Major V. M. Coppleson and Captain C. G. McDonald, Reserve of Officers, 2nd Military District, 1st July, 1940; and Frederick John Clark, 1st July, 1940; Alfred Charles Thomas, 22nd May, 1940. *To be Majors*—Lieutenant-Colonel A. H. Powell, Unattached List, 2nd Military District, 22nd May, 1940; Major R. M. W. Webster, Australian Army Medical Corps, 6th Military District, 1st July, 1940; Major (Temporary Lieutenant-Colonel) W. Park, Australian Army Medical Corps, 1st Military District, 22nd May, 1940; Major G. C. Burston, Australian Army Medical Corps, 3rd Military District, 1st July, 1940; Major (Temporary Lieutenant-Colonel) G. G. L. Stening, Majors R. J. Stabback, Australian Army Medical Corps, 2nd Military District, 22nd May, 1940, and 1st July, 1940, respectively; N. Eadie, Australian Army Medical Corps, 3rd Military District, 30th May, 1940; J. E. Gillespie, Australian Army Medical Corps, 3rd Military District, 4th May, 1940; R. J. A. Henderson, Australian Army Medical Corps, 3rd Military District, 1st July, 1940; K. W. Starr, Australian Army Medical Corps, 2nd Military District, A. L. Dawkins, Australian Army Medical Corps, 5th Military District, 10th May, 1940; K. S. Richardson, Australian Army Medical Corps, 2nd Military District, 1st July, 1940; J. M. Dwyer and H. M. Fisher, Australian Army Medical Corps, 4th Military District, 6th May, 1940, and 10th May, 1940, respectively; Captains (Temporary Majors) S. Plowman and R. N. Howard, Australian Army Medical Corps, 3rd Military District, 18th May, 1940, and 1st July, 1940, respectively; N. L. Sherwood, Australian Army Medical Corps, 1st Military District, H. M. Trethowan, Australian Army Medical Corps, 5th Military District, E. C. Palmer, Australian Army Medical Corps, 2nd Military District, 1st July, 1940; C. A. M. Renou, Australian Army Medical Corps, 3rd Military District, 14th May, 1940; O. E. Nothling, Australian Army Medical Corps,

1st Military District, R. B. Maynard, Australian Army Medical Corps, 3rd Military District, 1st July, 1940; W. E. Langford, Australian Army Medical Corps, 1st Military District, 23rd May, 1940; F. K. Mugford, Australian Army Medical Corps, 4th Military District, 10th May, 1940; Captain A. D. Byrne, Australian Army Medical Corps, 4th Military District, 21st May, 1940; Captains (Temporary Majors) R. T. Binns, Australian Army Medical Corps, 4th Military District, 1st July, 1940; J. S. Crakanthorp and F. W. Niesche, Australian Army Medical Corps, 2nd Military District, 7th May, 1940, and 1st July, 1940, respectively; Captains J. G. G. White, Australian Army Medical Corps, and R. McK. Rome, Australian Army Medical Corps, 3rd Military District, 4th June, 1940, and 18th May, 1940, respectively; Major C. W. Nye, Australian Army Veterinary Corps, 3rd Military District, with regimental seniority next after Major R. McK. Rome, 18th May, 1940; Captain A. D. Matheson, Australian Army Medical Corps, 3rd Military District, 16th May, 1940; Captains (Temporary Majors) D. A. Cameron, Australian Army Medical Corps, 1st Military District, 22nd May, 1940; F. N. Chenhall, Australian Army Medical Corps, 2nd Military District, 7th May, 1940; Captains J. D. Palandri, Australian Army Medical Corps, 5th Military District, 10th May, 1940; A. F. Hobson, Australian Army Medical Corps, 2nd Military District, 7th May, 1940; F. K. Wallace, Reserve of Officers, 5th Military District, 10th May, 1940; Major W. P. MacCallum, Reserve of Officers, 2nd Military District, 17th May, 1940; Major O. E. J. Murphy and Honorary Captain A. W. L. Row, Reserve of Officers, 1st Military District, 22nd May, 1940; Honorary Captains H. M. Cutler, Reserve of Officers, 2nd Military District, 22nd May, 1940; D. J. Thomas and D. O. Brown, Reserve of Officers, 3rd Military District, 28th May, 1940; Majors H. Rayson, Reserve of Officers, 2nd Military District, and E. F. West, Reserve of Officers, 4th Military District, Honorary Major J. B. Hamilton, Reserve of Officers, 6th Military District, and Captains A. J. G. Mackay and C. H. Osborn, Reserve of Officers, 3rd Military District, 1st July, 1940; George Bruce Dalrymple Hall, Richmond Jeremy, Stuart Vance Marshall, William Laurence MacDonald, Kerrod Bromley Voss, Maurice Phillip Susman and George Norman Lorimer, 1st July, 1940; Angus Johnston Murray, John Howell Halliday, Kenneth Beeson Noad, Edgar Frederick Thomson, Reginald Henshall Brindley Bettington, Clifford Stirling Colvin, 22nd May, 1940; and Edward Ford, 1st June, 1940. *To be Captains*—Captains (Temporary Majors) J. E. Sewell and J. F. Akeroyd, Australian Army Medical Corps, 3rd Military District, 1st July, 1940; J. Kingsley, Australian Army Medical Corps, 2nd Military District, 20th May, 1940; Captains S. W. Bryan, Australian Army Medical Corps, 2nd Military District, 15th May, 1940; C. W. K. Hardy and J. F. J. Cade, Australian Army Medical Corps, 3rd Military District, J. S. Chalmers, Australian Army Medical Corps, 6th Military District, 1st July, 1940; T. L. Tyrer, Australian Army Medical Corps, 3rd Military District, 30th May, 1940; J. M. Blair, Australian Army Medical Corps, 3rd Military District, Captain (Temporary Major) I. H. Sender, Australian Army Medical Corps, 2nd Military District, 28th May, 1940; Captains T. U. Ley, Australian Army Medical Corps, 3rd Military District, 24th May, 1940; R. J. Wheeler, Australian Army Medical Corps, 5th Military District, 1st July, 1940; S. L. Seymour, Australian Army Medical Corps, 4th Military District, 1st June, 1940; J. Ray, Australian Army Medical Corps, 3rd Military District, 18th May, 1940; R. J. Humphery, Australian Army Medical Corps, 2nd Military District, 15th May, 1940; R. Warden, Australian Army Medical Corps, 3rd Military District, 1st June, 1940; W. M. Irwin, Australian Army Medical Corps, 4th Military District, 10th May, 1940; W. K. Myers, Australian Army Medical Corps, 2nd Military District, 22nd May, 1940; F. Vickery, Australian Army Medical Corps, 2nd Military District, 7th May, 1940; V. G. Bulteau, Australian Army Medical Corps, 2nd Military District, 28th May, 1940; A. R. Hazelton, Australian Army Medical Corps, 2nd Military District, 1st July, 1940; R. J. Hoy, Australian Army Medical Corps, 1st Military District, 28th May, 1940; D. G. Perrett, Australian Army Medical Corps, 2nd Military District, 22nd May, 1940; J. R. Magarey, Australian Army Medical Corps, 4th Military District, 10th May, 1940; A. A. Moon, Australian Army Medical Corps, 2nd Military District, 7th May, 1940; J. L. Holme and A. M. Johnson, Australian Army Medical Corps, 2nd Military District, 28th May, 1940; B. L. Hellings, Australian Army Medical Corps, 3rd Military District, 30th May, 1940; I. T. Cameron, Australian Army Medical Corps, 3rd Military District, 1st July, 1940; J. R. Goding, Australian Army Medical Corps, 3rd Military District, 15th May, 1940; J. B. Devine, Australian Army Medical Corps, 3rd Military District, 28th May, 1940; J. J. Searby, Australian Army Medical Corps, 3rd Military District, 1st July, 1940; D. R. C. Wilson, Australian Army Medical Corps, 5th Military District, 22nd May, 1940; N. J. Bonnin, Australian Army Medical Corps, 4th Military District, 10th May, 1940; H. F. G. MacDonald, Australian Army Medical Corps, 3rd Military District, 18th May, 1940; R. F. West, Australian Army Medical Corps, 4th Military District, 21st May, 1940; T. H. Ackland, Australian Army Medical Corps, 3rd Military District, 14th May, 1940; B. Moore and J. R. Radcliff, Australian Army Medical Corps, 2nd Military District, 1st June, 1940, and 15th May, 1940, respectively; A. D. A. Mayes and R. R. Winton, Australian Army Medical Corps, 1st Military District, 1st July, 1940, and 1st June, 1940, respectively; R. E. Maffey, Australian Army Medical Corps, 2nd Military District, 1st July, 1940; J. O. Lavarack, Australian Army Medical Corps, 3rd Military District, 18th May, 1940; M. S. Alexander, Australian Army Medical Corps, 2nd Military District, 7th May, 1940; R. S. Wilkinson, Australian Army Medical Corps, 4th Military District, 10th May, 1940; H. O. G. Selle, Australian Army Medical Corps, 2nd Military District, 7th May, 1940; R. G. B. Cameron and J. I. Loewenthal, Australian Army Medical Corps, 2nd Military District, 1st July, 1940; W. S. L. Stening, Australian Army Medical Corps, 2nd Military District, 15th May, 1940; E. V. Barling, Australian Army Medical Corps, 2nd Military District, 18th May, 1940; D. M. Yeates, Australian Army Medical Corps, 1st Military District, 22nd May, 1940; K. B. Burnside, Australian Army Medical Corps, 3rd Military District, 1st July, 1940; G. C. H. Hogg and E. P. Row, Australian Army Medical Corps, 1st Military District, 22nd May, 1940, and 30th June, 1940, respectively; J. F. Sullivan, Australian Army Medical Corps, 2nd Military District, 15th May, 1940; C. H. W. Lawes and W. Freeborn, M.M., Australian Army Medical Corps, 2nd Military District, R. Motteram and P. C. R. Goode, Australian Army Medical Corps, 4th Military District, 1st July, 1940; Honorary Captains F. J. Fenner, Reserve of Officers, 4th Military District, M. Mayrhofer, D. W. MacPherson, B. S. Gillett and A. J. King, Reserve of Officers, 5th Military District, 10th May, 1940; Z. Schwartz and J. C. Stewart, Reserve of Officers, 3rd Military District, 14th May, 1940; W. H. Long, Reserve of Officers, 3rd Military District, 18th May, 1940; Captain D. G. Croll, Reserve of Officers, 1st Military District, 23rd May, 1940; Honorary Captain L. G. Morton, Reserve of Officers, 3rd Military District, 24th May, 1940; Captain J. E. Clarke, Australian Army Medical Corps, 3rd Military District, 1st July, 1940; Honorary Captain H. B. Woolford, Reserve of Officers, 2nd Military District, 28th May, 1940; Captains P. I. A. Hendry and S. E. J. Robertson, Australian Army Medical Corps, 2nd Military District, 1st July, 1940; Honorary Captain M. T. Cockburn, Reserve of Officers, 4th Military District, Captain L. W. Johnston, Reserve of Officers, 3rd Military District, Honorary Captain J. K. Harbison, Reserve of Officers, 2nd Military District, Captains B. M. Carruthers, Reserve of Officers, 6th Military District, and B. K. Rank, Reserve of Officers, 3rd Military District, Honorary Captains C. D. Jermyn, Reserve of Officers, 1st Military District, A. H. Lendon, Reserve of Officers, 4th Military District, 1st June, 1940; Captain L. A. Atkins, Australian Army Medical Corps, 2nd Military District, 6th June, 1940; Honorary Captains P. J. Parsons, Reserve of Officers, 3rd Military District, 25th June, 1940; H. I. Turnbull, Reserve of Officers, 2nd Military District, G. V. Smith, Reserve of Officers, 4th Military District, J. F. Park, Reserve of Officers, 3rd Military District, M. R. Gold, Reserve of Officers, 4th Military District, D. G. Picone and A. J. Kennedy, Reserve of Officers, 1st Military District, P. G. Hayes, Reserve of Officers, 3rd Military District, and A. Fryberg, Reserve of Officers, 1st Military District, 1st July, 1940; William Douglas Sturrock, 7th May, 1940; Alexander George Campbell Budge, 13th May, 1940; Donald Grant Duffy, Henry Devenish Steward, John Victor Latham, Stanley Jack Marcus Goulston, 15th May, 1940; Alexander Hugh McGregor, 18th May, 1940; Frank Edward Gallash, Arthur Bernard Sullivan, Charles Ruthven Bickerton Blackburn, David Norrie Fleming Leake, Walter Gerald Holt, Innes Albert Brodziak, Louis Samuel Loewenthal, Eric Hyman Goulston, 22nd May, 1940; Donald Blanchard Wherett, 23rd May, 1940; Kenneth Fitzpatrick Russell, 25th May, 1940; Norman Richard Wyndham, Norman Henry Rose, 28th May, 1940; Alex Keith Barrett, 1st July, 1940; Geoffrey Vernon Mutton, George Vincent Rudd, Arthur Liddon Webb, John Patrick Horan, Alexander John Marum Sinclair, John William McNamara, 1st June, 1940; James Cameron English, 15th June, 1940; William Aitken, 20th June, 1940; Thomas Heron Steel, 24th June, 1940; Robert Sydney Day, Ian Lovell Duncan, Richard Lloyd Cahill, Roderick Lionel Jeffrey, Robert Bradley Speirs, Gerald Carew Moss, David Floyd Mercer, Edward Winchester Levings, Bruce Hunter Anderson, John Frederick Clair Camphin Cobley, Hugo MacCartney De Burgh, Ernest Benjamin Jones, George Clifton Halliday, Hugh Severin Stephens, Henry Oliver Lancaster, Andrew Parkes Findlay, Roy Markham Mills and Richard Harpur Formby, 1st July, 1940.

3rd and 4th Reinforcements.—To be Captains—Captains E. W. Kyle, Australian Army Medical Corps, 5th Military District, 30th May, 1940; and K. J. Friend, Australian Army Medical Corps, 2nd Military District, 1st June, 1940; James Montague Bonnin, 1st May, 1940; and Donald Sutherland Brandt and Reginald Thomas Dalton, 1st June, 1940.

Australian Military Forces.

NORTHERN COMMAND.

First Military District.

Australian Army Medical Corps.

Honorary Captain J. L. Selwood is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 1st June, 1940, and to be Major (temporarily), 3rd June, 1940; Honorary Captains M. G. F. Donnan, H. W. Noble and R. A. Douglas are appointed from the Reserve of Officers (A.A.M.C.), and to be Captains (provisionally), supernumerary to establishment pending absorption, 16th August, 1940; Captains (provisionally) G. R. Kurrle and K. H. Hooper are brought on the authorized establishment, 10th August, 1940, and 26th August, 1940, respectively.

Australian Army Medical Corps Reserve.

To be Honorary Captain—Joseph Baynes Cribb, 19th June, 1940. (This cancels the notification respecting this officer which appeared in Executive Minute No. 154/1940, promulgated in *Commonwealth Gazette* No. 170 of 22nd August, 1940.)

To be Honorary Captains—Reuben Hertzberg, 7th August, 1940; Harry Green and Mervyn Powell, 16th August, 1940, and Alan Turner Paul Pitt, 21st August, 1940.

EASTERN COMMAND.

Australian Army Medical Corps.

Captain J. C. Loxton is transferred to the Reserve of Officers (A.A.M.C.), 19th June, 1940. The provisional appointment of Captain J. Davis is confirmed.

To be Captain (provisionally)—Alan Victor Day, 6th August, 1940. Major C. R. Cole is appointed to command the 2nd Casualty Clearing Station, Eastern Command, and to be Lieutenant-Colonel (temporarily), 1st May, 1940, *vice* Lieutenant-Colonel (temporarily) G. G. L. Stening, seconded to the A.I.F. Captain W. L. Corlis is transferred to the Reserve of Officers (A.A.M.C.), 9th August, 1940. Major S. L. Spencer is retired, 1st August, 1940. The resignation of Captain (provisionally) H. H. Webber of his commission is accepted, 10th August, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Stephen Graham Sandes and Geoffrey Langford Howe, 2nd August, 1940, and 13th August, 1940, respectively.

SOUTHERN COMMAND.

Third Military District.

Australian Army Medical Corps.

The appointment of Captain S. L. Townsend is terminated, 31st May, 1940.

To be Major (temporarily)—Captain (provisionally) J. B. D. Galbraith, 31st August, 1940.

Australian Army Medical Corps Reserve.

The resignation of Honorary Captain H. A. A. Altmann of his commission is accepted.

Fourth Military District.

Australian Army Medical Corps Reserve.

The resignation of Major R. M. Glynn of his commission is accepted.

Sixth Military District.

Australian Army Medical Corps.

The provisional appointments of Captains R. A. Lewis and C. A. Duncan are confirmed; the provisional appointment of Captain J. C. Fulton is terminated.

WESTERN COMMAND.

Australian Army Medical Corps.

Honorary Major F. J. Clark is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 2nd September, 1940.

To be Captain (provisionally)—Harry Leigh Cook, 9th July, 1940. Honorary Captain B. O. Bladen is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 15th August, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Lieutenant—Frank Rowe, 12th August, 1940.

Royal Australian Air Force.

Citizen Air Force: Medical Branch.

The following are granted commissions on probation with the rank of Flight Lieutenant, with effect from 2nd September, 1940: Derek Pelham Sapsford, M.B., B.S., and Edward George Strahan, M.B., B.S.

The following are granted commissions on probation, with the rank of Flight Lieutenant, with effect from the dates indicated: Hans Albert Adolph Altmann, M.B., B.S., Edward Campbell, B.D.Sc., John Henry Deakin, M.B., B.S., William Brien Marsh, M.B., B.S., Robert Gordon Mackay, M.B., B.S., William Peter McLaughlin, M.B., B.S., and Geoffrey Lawrence Young, L.M.S.S.A. (London), 19th August, 1940; David Barry, M.B., B.S., and Raymond Lloyd-Jones, M.B., B.S., 26th August, 1940.

Reserve: Medical Branch.

John Walter Broughton, M.B., Ch.M., is granted a commission on probation, with the rank of Flight Lieutenant, with effect from 12th August, 1940.—(Ex. Min. No. 68—Approved 25th September, 1940.)

Correspondence.

OBSTETRICAL PRACTICE IN BRISBANE.

Sir: Might I make comment on some aspects of obstetrical practice in Brisbane?

In Brisbane it appears there is a widespread system of vigorous interference as a policy in applied obstetrics. This is apparent from the attitude to their cases of nurses in private and public hospitals.

As an example it seems that parturient women are examined internally as a routine on admission, so that along with the information that one's case has arrived in hospital comes news of the condition of her cervix expressed in monetary or digital units.

Again, if the second stage of labour is rapid, one arrives to find a nurse repelling the head by force to prevent its untimely delivery, and another administering chloroform to still the hasty uterus. They are instructed, they say, not to take the responsibility of delivery before the doctor's arrival in intermediate or private cases. But rather are they encouraged to take the responsibility of holding back the head and administering a dangerous anæsthetic over a period of twenty or thirty minutes.

Is this modern scientific obstetrics? Are the interests of the patient best served by systems which have scant regard for the sanctity of Nature's way?

A doctor's and a nurse's duty is to ease pain, prevent and treat the abnormal, not to perform a hocus-pocus ritual for the sake of medical prestige.

Let us turn and view the ancient adage *"Primum non nocere"*.

Yours, etc.,

146, Yabba Street,
Ascot,
Brisbane.
September 6, 1940.

John K. Mowat.

PSYCHIATRY FOR THE GENERAL PRACTITIONER.

Sir: I have read with much interest the special articles on psychiatry which have been appearing in the journal. Some of them have been bright, very bright, even skittish—"To think of a skittish article on the psychoses", as Loudon Dodd doesn't say in "The Wrecker"; some—well, not so bright; however, what puzzles me is for whom they are intended.

Apparently not for the psychiatrist, for they are not full enough for him, presumably then for the man in general practice and yet does one general practitioner in a hundred read them or supposing he does read them, does he understand them, much less learn from them.

After all, mental diseases above all others are the ones where close personal association with cases is essential if one is going to learn anything of their natural history.

Anyway what does the average general practitioner do when confronted with mental disease? He either certifies the patient right away or calls in a specialist.

What can the general practitioner tell the relations when they ask him: (i) What mental disease has the patient got? (ii) What is the cause of it? (iii) Is he going to get better? (iv) Will he be violent or suicidal? (v) If he gets over it, will he get attacks later on? Mighty little, even the professional psychiatrist cannot say a great deal.

Now let us examine one of the articles, that on epilepsy, more closely.

When a patient is brought to the general practitioner, with a history of "fits", what is the first thing to do? Surely to make certain the "fits" are genuine epileptic ones and not attacks associated with some other disease; what

help does this article give on this point? Again, mighty little.

What is the next thing? Surely treatment, but why expensive proprietary drugs like "Luminal", "Prominal", "Luminal Sodium", when general experience has shown they are no better than the very much cheaper *Phenobarbitonum Solubile* of the British Pharmacopœia which is not even mentioned; furthermore, one might mention that a single dose of *Phenobarbitonum Solubile* given the first thing in the morning is, generally speaking, better than divided doses during the day; that solutions of any of these drugs soon deteriorate.

"A good nurse or sometimes a relative" knows well that next to the administration of sedatives, the regular action of the bowels is the most important part of treatment and yet no mention of it!

Now, about *status epilepticus*: first of all, if every epileptic who has two or three fits close together is given a copious enema of soap and water, the risk of *status epilepticus* is much reduced; apparently the copious enema acts as does colonic irrigation in eclampsia; if the fits do persist or *status epilepticus* sets in, give a rectal injection of one ounce of paraldehyde in one ounce of olive oil.

If I were asked to write an article about mental disease for the general practitioner, I should choose "depression", pointing out that it is not "neurasthenia", nor "nerves", nor "being run-down", but melancholia; that it runs a self-limited course and is not cured by the removal of teeth, tonsils nor appendix; that a mild case is more likely to commit suicide than a severe one; that suicide comes more often early or late in the disease and not at its height; that to send one of these patients on a sea voyage is inviting trouble.

Finally a tip to the general practitioner who has some familiarity with mental disease—never diagnose a case in an adult without a Wassermann test or sooner or later you will fall in.

Yours, etc.,

J. E. F. McDONALD.

Medical Superintendent, Toowoomba Mental Hospital, Willowburn; Visiting Medical Officer, Home for Epileptics, Rockville.

September 13, 1940.

KEEPING THE EYES ON THE BALL.

SIR: At games in which a swiftly moving ball is watched and struck by the player with a bat or racket, skill is largely a matter of the efficiency of the extraocular muscles. The ball must be followed throughout its flight and the spatial relationship of it accurately known in order that the player may strike with precision. Appreciation of correct spatial relationship relies on binocular stereoscopic vision, which is governed by coordination, speed of action and absence of fatigue of the extraocular muscles. This applies particularly in relation to a moving object, and the nearer the approach of the ball the greater is the muscular effort, especially of the medial recti, in maintaining binocular vision and serving convergence.

It will be helpful at this stage to imagine a triangular plane, the base being formed by the line joining the centre of the two eyes and the sides by the visual axes. The apex of the triangle is the point of central binocular gaze and the spatial relationship of any object at this point can be judged, judgement becoming more accurate the closer the object approaches the observer. Therefore, to keep accurately the eyes on a ball in flight the ball must be held, so to speak, at the apex of a mobile triangle. The importance of convergence as the main factor in this effort cannot be overstressed. The sum of the angles of convergence of each eye and the total muscular effort required to view an object at any given distance remains constant for any position of the head, so that we need not complicate the discussion by having to consider positional factors.

If there is a lag in convergence, the ball will fall inside the triangle, being then in the area of physiological heterogenous diplopia. Again, if the eyes converge more rapidly than the ball approaches it will fall outside the triangle, in the area of homogenous physiological diplopia. In each case judgement of the position of the ball will be faulty because it is no longer under binocular control, and the attempted stroke will fail. The bulk of the task of maintaining binocular vision falls on the convergers, the medial recti, and a measure of their efficiency is a measure of a player's skill, other factors being normal.

There are three main groups of players; firstly those who maintain a high skill over a long period, secondly those who begin brilliantly but show more or less rapid falling off, and those who are poor players throughout. Grouping of players according to their convergence efficiency, measured by orthoptics, follows closely the above. Players belonging to the first group show normal or greater than normal convergence and orthophoria, and have the greater sustained skill; those with poor convergence and/or heterophoria, have little or no skill and belong to the third group. The second group is intermediate and, as play progresses, reveals more or less gradual loss of convergence efficiency, which is due to fatigue of the ocular muscles, and skill diminishes accordingly.

These remarks, which are based on a number of observations but actually too few to tabulate, will open up a field for further investigation. There is also the possibility of improving a player's batting average or his skill at tennis by orthoptic training, especially, I believe, does this apply to the second group, although a number of the third group may be lifted out of mediocrity. Where the ball is in a fixed position, as at golf, the importance of control and coordination of the ocular muscles may also be emphasized.

Keeping the eye on the ball has greater significance if we analyse the main ocular factors involved and their bearing on the player's skill. The traditional advice to "keep your eye on the ball" would be more accurate in the plural, "keep your eyes on the ball".

Yours, etc.,

JOHN MAUDE.

British Medical Association House,
135, Macquarie Street,
Sydney.

September 19, 1940.

THE PUBLIC AND NUTRITION.

SIR: In listening last night to the British Medical Association Spokesman's broadcast upon nutrition, I could not help feeling that its undoubted truth and excellence would have been less ironically vitiated if it had been made clear that he was addressing only those with incomes of at least ten pounds a week. The advice which he gave, sound as it was, cannot, obviously, be offered with any sincerity to an audience many of whom would be on the basic wage or the 7s. 6d. dole. To such people an address of this kind is not helpful; it is merely insulting. It contained not a sentence, not a word, to suggest that in New South Wales there are scores of thousands of people far too poor to buy the butter, cheese, milk, cream, green vegetables, eggs and fruit, the need for which was stressed again and again.

It should not be difficult, surely, to imagine the thoughts of an unemployed listener who, with his wife and children, is existing on food relief. He has watched his family being underfed for years; he knows quite as well as the Sociological Department does that there is "plenty of food in Australia", and, having heard that there is to be a talk on nutrition, sponsored by the British Medical Association, he hopes to hear something said about the urgent need for a more equitable distribution of that plenty. Perhaps he even hopes to hear a protest which may have some chance of being heeded, coming as it does from a body with the knowledge, power and prestige of the British Medical Association. But "the hungry sheep look up, and are not fed".

With the possible exception of war, poverty is the greatest cause of disease and death in the world; thus it becomes, inescapably, a medical problem. As an indication of its tragic effect, consider these figures from the English Registrar-General: recently infant mortality for the third and fourth trimesters among the independent class was 32‰, but 137‰ among the labouring classes.

There can be no question of adequate nutrition for those on the dole. It is impossible, and it is surely the duty of the Sociological Department to say so, and to follow out all the implications. I also suggest that another very necessary work for this body is to investigate the possibility of full nutrition for workers on the basic wage, taking into account all the expenses which must be met before food is bought—rent, clothing, light, heating, travelling to work, union dues, lodge fees, hospital subscription and dental expenses—and also assuring themselves that the rent allowed is the actual rent which would have to be paid for a house fit for a family consisting of man and wife and two children. Moreover, in calculating what food might be bought with the remainder of the week's wage, it should be remembered that it must be bought in small quantities, and mostly in little shops within walking distance of the home.

As the worker should not be expected to live by bread alone, a reasonable allowance should be added for culture and recreation, including at least a fortnight's holiday, and for smoking, as that has now become part of a normal person's life.

There are many other sociological investigations for the members of the department to make, and discuss publicly. If they act with energy, vision and courage it is certain that they could have a great influence on the life of this State.

Yours, etc.,

E. P. DARK.

Katoomba,
New South Wales,
September 26, 1940.

VASO-CONSTRICTORS AND SPINAL ANÆSTHESIA.

SIR: Dr. Morgan's address on spinal anæsthesia controlled to reach different body levels shows great practical achievement. Permit me to comment on the use of vaso-constrictors in spinal anæsthesia, basing my conclusions on 468 cases of my own with no mortality.

Vessels below the nipple line (the usual uppermost limit) relax as the splanchnic nerves are blocked. Vessels above this line are in their normal tone and offer greater resistance to the blood stream. These factors cause blood to "pool" below the nipple line, while the brain becomes anæmic. If epinephrin is given, the blood pressure in the arms may rise, owing to capillary contraction; but the splanchnic vessels remain unaltered, as stimuli cannot travel over the anæsthetized tracks. This results in the blood still further collecting in the splanchnic area and in a still smaller quantity of blood in the brain, with more pronounced cerebral anoxæmia.

I used vaso-constrictors for some three years, but fear their use. I now place reliance on two factors: (i) a sufficiently pronounced Trendelenburg position for gravity to help the filling of the cerebral vessels; (ii) a sufficient quantity of the anæsthetic drug.

Notes of three recent cases are given, in which operation was performed under spinal anæsthesia with "Novocain" and without premedication.

Mr. B. was aged seventy-eight years. The condition of his heart made him a very bad operative "risk"; he weighed twenty stone and he had a large strangulated umbilical hernia through the scar of a previous operation for hernia. At operation extensive adhesions were dealt with, a large mass of omentum and eight inches of lifeless bowel were resected, a lateral anastomosis of the jejunum was performed, and relaxation permitted careful repair of the wound. The patient was unconcerned throughout the proceedings; his condition permitted tranquil operating.

Baby A., aged nine months, was operated on on September 12, 1940, for total intussusception of the colon. When the incision was made, dilated small bowel and blood-stained serum escaped. Reduction was commenced within the abdomen till the splenic flexure was passed, and it was completed extraabdominally. The baby was asleep during the latter part of the operation and while the wound was being sutured.

Mrs. S., aged twenty-four years, was operated on on August 28, 1940; she had swallowed an ordinary pin two months previously. The patient had been kept under observation during this period. She had previously suffered from attacks of abdominal pain, chiefly in the central area. At operation the small bowel was examined from the caecum to the duodenum and the large bowel from the caecum to the sigmoid colon. It was found that chronic appendicitis was present, and the right margin of the omentum was adherent to the hepatic flexure of the colon. The bowel examination was repeated, as a loop of ileum had passed under the adhesion to the colon; the pin was noted in the rectum. The lesions were dealt with, the abdomen was closed and the pin was removed *per rectum*. The patient's pulse rate remained at 70 per minute and her colour good; she was speaking on different topics during the operation.

Yours, etc.,

E. A. JOSKE.

Balaklava,
South Australia,
September 19, 1940.

THE REORGANIZATION OF THE MEDICAL PROFESSION.

SIR: I would like through your columns to express my appreciation of the thoughtful and stimulating paper by Dr. Brown, of Colac, published in a recent issue of your journal. This paper merits the closest attention of every member of the profession who is in any way interested in the future of medical practice. At the present time, of course, our main objective is to win the war. But this should not prevent us from giving serious thought to the inevitable reconstruction that must follow, and this will involve medical practice as it will many other things. Up to now our democratic form of government has seemed to put a premium on inefficiency; but this must be altered.

To indulge in recrimination may not be a profitable business, but we should recall the words ascribed to Mr. Churchill: "The use of recrimination about the past is to enforce effective action at the present." It is necessary then to remember the pitiable fiasco of the attempt to introduce national insurance and of the abject failure of our Federal Council to do anything to protect the interests of the public or the profession. Some of the men responsible for that failure saw the light and resigned. Others were forced out of office. Others again, every whit as much to blame, still hold office. A failure as gross as that is unpardonable, and it is difficult to see how such men can expect to retain the confidence of the profession in any major scheme of reconstruction.

I have studied Dr. Brown's suggestions carefully and I approve them. But I would like to hear more details. There will be difficulties, but these can be overcome. The public looks to the profession for a lead, and we cannot afford to fail them again. Naturally these things want talking over and discussing fully; but I think the germ of truth and wisdom lies in Dr. Brown's ideas.

Although I hold no exalted opinion of our Federal Health authorities, I am in health matters an out-and-out unificationist. It seems to me an absurdity that there should be a different medical act for each State. If the Federal Health Department controls quarantine, why should it not control the whole of the health activities of the nation? Many times I have seen the position arise in which the State authority says a particular matter is the function of the Federal authority, and the Federal authority says it is a matter for the State. And between the two nothing is done. A glaring example is the 14s. grab from the pension of a tubercular patient who enters a sanatorium. Neither Government will accept responsibility for this absurdity. Why can't we have a general medical council on the lines of that in England, with the necessary added powers, to act as the central authority in all health matters. If distance is a bar there could be a small local authority in each State to act under its general jurisdiction. Such a body would be admirably adapted to administer a scheme such as that propounded by Dr. Brown. I hope medical men will put on their thinking caps, and try to evolve some workable scheme of their own, instead of having something that may be objectionable forced upon them by politicians who seem incapable of seeing anything except the financial side of the problem.

Yours, etc.,

D. R. W. COWAN.

172, North Terrace,
Adelaide,
September 28, 1940.

VENEREAL DISEASE AND THE ARMY.

SIR: I recently stated that the defeat of the Fifth Army in March, 1918, could be attributed to the number of men in all the British Armies absent owing to venereal disease. The statement was challenged and I cabled London (The British Social Hygiene Council) to ask whether I was correct in stating that during the Great War 400,000 men were infected. The reply was in the affirmative and that they were off duty for six weeks. I was referred to the "Official History of the War", Volume II, page 118.

In 1917 (see page 126) a letter was issued by the Army Council directing attention to the serious wastage resulting from venereal disease and to the new measures of prevention about to be adopted and holding commanding officers personally responsible for seeing that everything possible was done to make them a success.

The rate for the whole British Army, including Dominion troops, appears to have been about 5% *per annum*, which is probably not far off the figures in civil life, though we shall not know the extent in civil life until, as in the United States of America, a one-day census is taken in cities. The result has generally been round about 1% of the population on any one day.

In the "Official History" one remarkable fact is stated. A number of men suffering from syphilis died as a result of arsenical treatment which was accordingly modified.

I do not know the precise number of men who were disabled by venereal disease in March, 1918—I was then in Egypt—but I know it to be great.

It is obvious that two or three divisions in reserve could have stopped Ludendorf's rush and given time for reinforcement. As it was, 7,000 yards of the line were held by one

division against which Ludendorf threw several divisions with a general numerical superiority of three to one.

May I repeat that an efficient military medical service can render victory possible and a well-meaning but slack service can render defeat certain.

I may add that the "Official History" supported my views expressed in a work of mine, "A Vision of the Possible", that the management of venereal disease in an army requires a staff of specialists and a director.

Yours, etc.,

James W. Barrett.

103-105, Collins Street,
Melbourne,
September 30, 1940.

WAR WOUNDS AND THEIR TREATMENT.

Sir: Your leading article (September 28, 1940) betrays an anxiety state which seems also to pervade the medical journals which you enumerate. I have perused all these and still wonder to what extent the younger medical officers will benefit trying to digest them. It would be unfortunate for one of them to work under the eyes of a senior whose views did not coincide with the measures the junior had selected from his reading. No definite standard of treatment for all classes of wounds has yet emerged from the mass of lectures and literature which has confronted us for years past. The problem of splinting simple fractures was settled in no time in the Great War by the late Sir Robert Jones, a reduction of the complex to simplicity. Would that the minds of men now employed in the care of the wounded would turn to the standardized simplicity of the Listerian way. To do so they must learn that the most important duty is the prevention of infection and not the cure. Up to the present the attempts made at prevention are futile, thus bringing into prominence means of cure or prophylaxis of an impracticable nature. Lister never attempted to kill infection in a wound—a widespread erroneous idea—he relied on natural tissue resistance to infection to deal with that and gave all his attention to the prevention of secondary infection. An intelligently applied antiseptic dressing is portable and inexpensive and is all that is needed whether the wound be infected or not. Irrigation, *débridement*, gauze plugging are all harmful in the eyes of those who adopt the antiseptic principle.

Yours, etc.,

A. C. F. Halford.

Brisbane,
October 1, 1940.

OPTICAL CONVERGENCE AND STEREOPSIS IN RELATION TO PERSPECTIVE.

Sir: In his article in the journal of September 28, on "Optical Convergence and Stereopsis in Relation to Perspective", Dr. John Maude draws our attention to the relationship between optics and art. His argument is introduced by a short sketch of the evolution of stereoscopic vision, in which he summarizes the development of the highly efficient visual mechanism of the master creature, *Homo sapiens*, from the crude eyes and visual cells of the lower vertebrates. Unfortunately the facts revealed by a study of comparative anatomy do not agree with this facile explanation. On the contrary the appearance and development of the vertebrate eye is one of the great problems of comparative anatomy, and whilst one hesitates before criticizing the utterances of such an authority as Elliot Smith, it would appear from his writings that he was not aware of many of the findings of diligent research workers on the comparative anatomy of the vertebrate eye.

The macula is by no means the private property of the primates and man. It is present in a well-developed form in quite a number of fish, it is actually degenerating in the archaic reptile *Sphenodon*, it is found in every lizard with a round pupil and in two snakes, whilst many birds possess two in each eye. I have been amazed to find a deep fovea in some of the smallest of our lizards. For example, Guichenot's skink, with an eye less than three millimetres in length, has quite a deep *fovea centralis*. The eyes of mammals are poor in comparison, and it is very doubtful whether the *fovea centralis* of man is as efficient as that of a bird of prey. Lizards and birds, the majority of which are strongly diurnal, rely to a very large extent on their visual organs in their hunt for food. The lizard, indeed, has gone so far as to discard the rods from the retina. What degree of binocular vision is possessed by the sauropsidians is problematical, but I have often noticed in lizards and snakes that a reflex from the temporal fundus can be obtained with an ophthalmoscope held in a line directly ahead of the snout of the reptile. The foveæ of the snake *Dryophis* are in the temporal periphery, and are probably used in binocular vision, as are the temporal foveæ of birds. We cannot put them on the synoptophore to ascertain whether they are aware of a third dimension, although we are fully aware of their accuracy when striking down their prey.

It is impossible, in a short letter, to adduce all the facts which tend to disprove the popular theory that the efficiency of the visual apparatus improves with the ascent of the phylogenetic scale. The lens of the fish is globular, not because the fish's eye is a crude optical instrument, but because a strongly refracting lens is necessary to compensate for the loss of refraction between the surrounding water and the corneal surface. Matthiessen proved long ago that the optical constants of the fish's eye are remarkably accurate and uniform from species to species. The nocturnal gecko has a larger cornea and lens than his diurnal cousins the skinks and the "goannas". The crepuscular bandicoot has a smaller cornea and lens than his more highly developed relative—the very nocturnal ring-tailed opossum. And what are we to say of the bats, whose position in the phylogenetic scale is very high indeed? It would seem that the structure of the eye depends rather on habit than phylogeny.

Helmholtz, in one of his popular scientific lectures, discussed the subject of optics and painting and allowed much more to the one-eyed man than parallax to help him in the perception of depth. However, he did not have the help of the modern improvements to the stereoscope and was much less trusting in the infallibility of his instruments than is the modern investigator.

In conclusion I should like to thank Dr. Maude for his very unusual paper. Specialist practice tends to become very stereotyped, and an essay such as his reminds us that we are not entirely divorced from the arts.

Yours, etc.,

Kevin O'Day.

33, Collins Street,
Melbourne, C.1,
October 1, 1940.

THE INTERACTION OF MIND AND BODY.

Sir: I should like to congratulate Dr. Harold Ritchie on his brilliant and lucid Anne MacKenzie oration, dealing with the vitally important subject of the interaction of mind and body. Like Dr. Ritchie, I believe that this problem is one which will receive much attention in the next chapter of the history of medicine; and I also think that here and now it should be receiving a great deal more attention than it actually is.

The importance of the influence of psychological factors in illness has been recognized for a very long time, but it is only quite recently that the physician is beginning to accept the fact seriously and attempt to do something about it. One would be encouraged to believe from Dr. Ritchie's address, and from the amount of general interest in this subject that the present curriculum of students would naturally provide for a future supply of graduates adequately trained in medical psychology. But our training is still definitely backward in this direction, and one has to try to teach clinical psychological medicine to students who have had no preliminary lectures in psychology. It is none the less probable, almost certain in fact, that the growing recognition of the interaction of mind and body in the ætiology of disease will soon require an uncomfortable amount of readjustment in our approach to medicine. The emerging facts will have to be faced eventually and a great many established theories will go into the melting pot. Perhaps some of the more rigid-minded leaders of our profession are unconsciously putting off the evil day of readjustment by trying to keep psychology down.

However this may be, medical psychology is sufficiently advanced and established today to make it essential that every graduate should be turned out with an awareness of the psychological as well as of the physical and biological approach to medicine. He should also be convinced that there is always present in illness a factor which is not fully explained in his text-books.

There is one other point which I should like to mention. Dr. Ritchie suggests that in many cases it is only necessary to detect the patient's conflict and confront him with it to relieve his symptoms. Unfortunately in practice this does not happen nearly as often as one would wish. It seems that when improvement does occur in such a case it is usually only temporary, depending on the degree of emotional release which accompanies intellectual acceptance. Generally speaking, to tell an ostrich with his head in the sand that he is plainly visible to his pursuers is not likely to be enough. He will probably react in one of the following ways, either by refusing to believe you because he cannot see and leaving his head safely buried, or else, reassured by your

friendly voice, he might withdraw his head, take a quick look round, and realizing his danger afresh thrust his head in more deeply than ever or perhaps, in this case, he would run hard in another direction and bury it again somewhere else. For his real equanimity we must not remove his head from the sand until we have convinced him that his pursuer (that is, his own anxiety) is not really dangerous and will not destroy him if he looks up and faces it squarely.

I think that Dr. Ritchie expresses a profound truth when he says that "the problems of adaptation to environment are the background to half the ills the flesh is heir to".

Yours, etc.,

CEDRIC SWANTON.

British Medical Association House,
135, Macquarie Street,
Sydney.
October 2, 1940.

SPRING CATARRH.

SIR: As promised, I beg to report on the case of spring catarrh previously referred to in THE MEDICAL JOURNAL OF AUSTRALIA and treated by local application of "Prontosil Soluble" 2·5%.

As anticipated, with the spring there has been a slight recurrence, but it is well in hand and the patient has complete comfort.

The treatment has been varied by hypodermic injection of the drug into the conjunctiva at intervals.

Whilst complete cure cannot be claimed, there is marked improvement and the thickening in the worse eye has so much reduced that it is difficult to get the hypodermic needle into it.

Yours, etc.,

JAMES W. BARRETT.

103-105, Collins Street,
Melbourne, C.1,
October 2, 1940.

PINK DISEASE TREATED WITH VITAMIN B_1.

SIR: In commenting on Dr. Forsyth's reports in the journal (November 18, 1939, and March 30, 1940) of the successful use of "Bemax" (one tablespoonful *t.d.s.*) in the treatment of pink disease, Dr. Douglas Wright suggests that the effect might be due to vitamin *E*.

While it is of course true that "Bemax" has a very high vitamin *E* content (approximately eight milligrammes of α-tocopherol per ounce, that is 12 milligrammes in the dosage used by Dr. Forsyth) it is perhaps relevant to point out that deficiencies of two of the other vitamins present in "Bemax" (B_1, 400 international units per ounce; B_6, no units available, but wheat germ is stated by Birch, György and Harris to be the richest source[1]) both play fundamental roles in the production of diseases of the nervous system.[2]

From the economic aspect the concentration of these three vitamins, B_1, B_6 and *E*, is so high in "Bemax" that the best interests of the patient would probably be better served by giving all these vitamins as they occur naturally in "Bemax" rather than by substituting any one synthetic preparation which can only replace one of them at a cost three or four times that for which the three are provided in the natural substance.

This suggestion applies with particular force in the case of vitamin *E*, since the usual dosage of the synthetic vitamin—one tablet *t.d.s.*—would not provide the amount of vitamin *E* yielded by the dosage of "Bemax" used by Dr. Forsyth.

Yours, etc.,

H. C. H. GRAVES,
Director.

The Research Laboratories,
Vitamins Ltd.,
23, Upper Mall,
Hammersmith, London, W.6.
August 8, 1940.

References.

(1) T. W. Birch, P. György and L. J. Harris: "Vitamin B_2 Complex", *The Biochemical Journal*, Volume XXIX, December, 1935, page 2830.

(2) W. Antopol and C. E. Schotland: "The Use of Vitamin B_6 in Pseudohypertrophic Muscular Dystrophy", *The Journal of the American Medical Association*, Volume CXIV, March 23, 1940, page 1058.

Obituary.

ARCHIBALD WATSON.

WE are indebted to Dr. Bronte Smeaton for the following account of the career of the late Professor Archibald Watson.

Archibald Watson began his medical studies later in life than is usual. He first took the Fellowship of the College of Surgeons in England, then later M.D. Paris, and M.D. Göttingen. While studying in Paris he was a contemporary of Pierre Marie, whose subsequent fame delighted Watson. Teaching in Cook's school of anatomy in London, he met Edward Stirling. When Sir Thomas Elder made an endowment to the University of Adelaide sufficient to establish a medical school, Stirling advised the appointment of Watson as professor of anatomy. He taught also pathology, surgical anatomy and operative surgery. He remained professor of anatomy from 1884 until 1919, when he was appointed emeritus professor of anatomy. Since his retirement many graduates who had been his pupils, and other admirers, subscribed a sum of money sufficient to obtain an excellent portrait, painted by McInnes—each subscriber received a replica of smaller size. [The portrait is reproduced herewith.] A sum remaining was devoted to the establishment of a Watson Prize at the university. Watson's will provided a legacy of £1,000 to be divided among those of his pupils who contributed; it has been suggested that means might be found to use this legacy (which will realize £800) for the endowment of a further Watson prize or scholarship at the University of Adelaide.

Watson was a teacher of no usual type. In vivid language, with clever, rapid sketches on the blackboard, he taught with a dramatic intensity that compelled the attention of his class. The anatomy he taught was largely surgical anatomy, and he would illustrate his subject with unqualified descriptions of the surgical triumphs and disasters of our clinical teachers and others. One felt that he allowed nothing to mar the dramatic necessities of the situation he described. Both in intellect and temperament Watson was many-sided. A great talker—with imagination—he made experiences or incidents live before one and there was often a flash from an unsuspected facet.

The Adelaide Hospital dispute, with the resulting depletion of the staff, gave him easy access to the operating theatre and quickened his interest in surgery. Watson's knowledge of anatomy gained in the dissecting room and in the post-mortem room enabled him to apply his wonderful mind to the problems of surgery, and his teachings were sought after by many surgeons in the Australian capitals and teaching schools.

The extent of his influence on surgery in Australia and elsewhere is difficult to exaggerate. He had a passion for the preservation of the tissues, and would denounce the unnecessary destruction of even the smallest subcutaneous vein. Watson had an unusual appreciation of the anatomical planes of the body and the possibilities they gave of a bloodless approach or mobilization of a viscus. He drew attention to, if he did not discover, the value of the division of the lateral blade of the mesentery of the colon as a means of mobilizing it. His anatomical knowledge of the blood supply of the uterus and his teaching that the vessels could be exposed by division of the peritoneum made hysterectomy a precise and safe operation, and clamps disappeared. Watson's teaching in this respect I have seen acclaimed in American literature. A great deal of the early work on hydatid disease was done by him and found its way into text-books.

He served in the Boer War as consulting surgeon to His Majesty's forces in Natal. He also served in Egypt during the Great War.

Outside of his teaching work his interests were innumerable and his industry intense. When quite an old man he made a wide study of electricity and its application. He sailed the seas over, observing the migrations of the tuna and other large fish. A great lover of the sea, he travelled widely in all the tropical and temperate seas and spoke the language of many countries.

In his passing we have lost a great man who never faltered in his search for scientific truth.

Many of us also have lost a friend unlike all others.

ALEXANDER STONEY PATTON.

WE regret to announce the death of Dr. Alexander Stoney Patton, which occurred on September 26, 1940, at sea.

Analytical Department.

"STOTT'S ÆTHER FOR ANÆSTHESIA".

"STOTT'S ÆTHER FOR ANÆSTHESIA" is manufactured by the Stott Chemical Company, Proprietary, Limited, Abercrombie Street, Sydney. A sample of this ether was submitted to our analyst, who furnishes the following report:

> We have examined the sample of "Stott's Æther for Anæsthesia" submitted by you for conformity with the tests for purity prescribed in the British Pharmacopœia (1932).
>
> The results obtained were as follows:
>
> | | |
> |---|---|
> | Specific gravity at 15·5°/15·5° C. .. | 0·720 |
> | Boiling point .. .. | 34°-35° C. |
> | Test for limit of sulphurous acid and other free acids .. .. | Sample conforms |
> | Residue on evaporation of 50 ml. .. | 0·0003 gramme |
> | Foreign odour on evaporation .. .. | None detectable |
> | Test for limit of peroxides .. .. .. | Sample conforms |
> | Test for limit of acetone and aldehyde | Sample conforms |
> | Test for limit of methyl alcohol .. .. | Sample conforms |
>
> *Storage*: The sample is contained in an amber bottle, fitted with tin-foil covered cork.
>
> The sample conforms in all respects with the requirements of the British Pharmacopœia specification.

We conclude that "Stott's Æther for Anæsthesia" may be recommended as a reliable ether for the use of medical practitioners in the practice of anæsthesia.

Notice.

LIBRARY SEMINAR AT SYDNEY.

THE next library seminar arranged by the Post-Graduate Directors of Medicine, Surgery and Pathology will be held at the Prince Henry Hospital, Little Bay, New South Wales, on Monday, October 14, 1940, at 4.30 p.m. The subjects will be: (*a*) "Unabsorbable Ligature Materials", (*b*) "Thrombosis". A cordial invitation to be present is extended to all medical practitioners.

Medical Appointments.

Dr. H. H. Stewart has been appointed a Member of the Nurses' Registration Board of Western Australia.

* * *

Dr. N. F. Babbage has been appointed Government Medical Officer at Emmaville, New South Wales.

* * *

Dr. H. A. Goode has been appointed Temporary Honorary Assistant Physician at the "Mareeba" Babies' Hospital, Adelaide.

* * *

Dr. P. E. Walton-Smith has been appointed Honorary Consulting Pathologist to the Royal North Shore Hospital of Sydney.

* * *

Dr. I. R. Horn has been authorized to sign permissions and certificates for cremation and to grant permission to cremate any human body after death, in accordance with the provisions of *The Cremation Acts*, 1913 *to* 1935, of Queensland.

* * *

The following appointments have been made at the Royal Adelaide Hospital, Adelaide, South Australia: Temporary Clinical Assistant to the Applied Physiology Department, Dr. J. D. Fotheringham; Honorary Clinical Assistant to the Medical Section, Dr. G. E. Peters; Temporary Honorary Clinical Assistant to the Ophthalmological Section, Dr. R. L. Naylor; Temporary Honorary Clinical Assistant to the Medical Section, Dr. R. K. Stockbridge.

Books Received.

"A Manual of Embryology. The Development of the Human Body", by J. E. Frazer, D.Sc., F.R.C.S.; Second Edition; 1940. London: Baillière, Tindall and Cox. Royal 8vo, pp. 533, with 282 illustrations. Price: 30s. net.

"Atlas of Cardio-Roentgenology", by H. Roesler, M.D., F.A.C.P.; 1940. Baltimore: C. C. Thomas. Crown folio, pp. 138. Price: $8.50 post paid.

"Surgery of the Hand", by R. M. Handfield-Jones, M.C., M.S., F.R.C.S.; 1940. Edinburgh: E. and S. Livingstone. Imperial 8vo, pp. 149, with 95 illustrations, some of which are in colour. Price: 15s. net.

Diary for the Month.

OCT. 15.—New South Wales Branch, B.M.A.: Ethics Committee.
OCT. 16.—Western Australian Branch, B.M.A.: Branch.
OCT. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 23.—Victorian Branch, B.M.A.: Council.
OCT. 24.—New South Wales Branch, B.M.A.: Clinical.
OCT. 25.—Tasmanian Branch, B.M.A.: Council.
OCT. 25.—Queensland Branch, B.M.A.: Council.
OCT. 31.—New South Wales Branch, B.M.A.: Branch.
NOV. 1.—Queensland Branch, B.M.A.: Branch.
NOV. 5.—New South Wales Branch, B.M.A.: Organization and Science Committee.
NOV. 6.—Western Australian Branch, B.M.A.: Council.
NOV. 6.—Victorian Branch, B.M.A.: Branch.
NOV. 7.—South Australian Branch, B.M.A.: Council.
NOV. 8.—Queensland Branch, B.M.A.: Council.
NOV. 12.—Tasmanian Branch, B.M.A.: Branch.
NOV. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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